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PHYSIOLOGIC AND PHYSICAL LAWS THAT GOVERN AUSCULTATION, AND THEIR CLINICAL APPLICATION

THE ACOUSTIC STETHOSCOPE AND THE ELECTRICAL AMPLIFYING STETHOSCOPE AND STETHOGRAPH

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I. Introduction

THE stethoscope is over 120 years old. It has withstood the criticisms of the earlier decades of its life, and its position as a diagnostic instrument is secure. The physical and acoustic principles of this instrument, however, have been imperfectly understood, and insufficient investigation has been undertaken in the analysis of the various stethoscope forms. In most instances, when such study has been attempted, the chest piece has been used as a sound collector of controlled vibrations in open air, and not in its normal position against the chest wall. In a sense, it has been compared to an airplane detector rather than a submarine detector.

At the Cardiac Clinic of the Massachusetts General Hospital, since its foundation by Dr. Paul D. White, in 1914, the importance of using both the bell and diaphragm chest pieces in examination of the heart has been insisted upon. The clinical work of one of us (H. B. S.) led, in 1925, to the combining of the two chest pieces, connected by a valve, into a single instrument. Other methods of combining them had been previously devised, but were not familiar to the profession in this country. Although they are clinically valuable, the exact acoustics of such instruments have not been previously analyzed by modern electrical methods. The engineering and manufacturing experience of one of us (M. R.) made it possible to undertake such a study. The investigation was extended to include ideal stethoscope forms, the influence of tubing length and material on the binaural stethoscope, diaphragm thickness, a comparison of binaural with monaural effects, types of heart sounds and murmurs, and energy output of cardiac vibrations. The study of the acoustic or mechanical stethoscope led to a review of the advances in

electrical transmission and recording of vibratory phenomena over the heart, and to a description of a new instrument for accurate registration of these phenomena by simultaneous employment of the electrocardiograph, stethograph, and pulse wave recorder.

We felt that the present interest in the graphic analysis and recording of the vibrations produced by the heart indicated a need for a more fundamental approach to the subject. The auditory phenomena of the heart can be understood only by the application of certain general principles of acoustics. These principles have been much more clearly defined in recent years by the electrical study of the human ear and by the perfection of electrical amplification.

We wish to express our appreciation of the cooperation of the Sanborn Company of Cambridge in this investigation. We are also grateful for the constructive criticisms of Professor Hallowell Davis of the Harvard Medical School and Dr. Paul D. White of the Massachusetts General Hospital.

II. History of the Stethoscope

Prestethoscope Era.—Auscultation, a method of investigation of the functions and conditions of the respiratory, circulatory, digestive, and other organs by the sounds they themselves produce, or that are elicited by percussion, is one of the most ancient modes of diagnosis. From the classic passage in Hippocrates' work, "de Morbis," "If you listen by applying the ear to the chest," we have definite evidence that "immediate" auscultation was employed more than twenty centuries ago.

Harvey's dissertation, "de Motu Cordis," gives convincing information that the sounds of the heart had not escaped him; "it is easy to see when a horse drinks, that water is drawn in and passed through the stomach, with each gulp, the movement making a sound, and the pulsation may be heard and felt. So it is with each movement of the heart when a portion of the blood is transferred from the veins to the arteries, that a pulse is made which may be heard in the chest."

Corvisart made the following observation: "Some authors assert that they could hear, in certain diseases of the heart, the noise produced by the violent strokes of this organ, even at a small distance from the patient's bed. I have never had an opportunity, I repeat it, of ascertaining the unquestionably rare observations; I have barely heard these strokes by applying my ear close to the patient's thorax."

However, during the prestethoscope era physicians derived little benefit from auscultation other than that of sensing the pulsations of the heart by the mere application of the hand.

The Laennec Stethoscope.—"I was consulted," says Laennec[1] (Traité de l'auscultation médiate, tome 1, p. 7), "in 1816 by a young woman

who presented some general symptoms of disease of the heart, in whose case the application of the hand and percussion gave but slight indications, on account of her corpulency. On account of the age and sex of the patient, the common modes of exploration being inapplicable, I was led to recollect a well known acoustic phenomenon, namely, if the ear be applied to one extremity of a beam, a person can, very distinctly, hear the scratching of a pin at the other end. I imagined this property of bodies might be made use of in the present case. I took a quire of paper which I rolled together as closely as possible, and applied one end to the precordial region; by placing my ear at the other end, I was agreeably surprised at hearing the pulsation of the heart much more clearly and distinctly than I had ever been able to do by the immediate application of the ear."

Laennec experimented further with his tube and finally evolved the instrument known as the "stethoscope."

"This consists simply of a cylinder of wood, a foot in length, perforated in its center longitudinally, by a bore three lines in diameter, and formed so as to come apart in the middle, for the benefit of being more easily carried. One extremity of the cylinder is hollowed out into the form of a funnel to the depth of an inch and a half, which cavity can be obliterated at pleasure, by a piece of wood so constructed as to fit it exactly, with the exception of the central bore which is continued throughout it, so as to render the instrument in all cases, a pervious tube. The complete instrument, that is, with the funnel-shaped plug infixed, is used in exploring the signs obtained through the medium of the voice and the action of the heart; the other modification, or with the stopper removed, is for examining the sounds communicated by respiration. A solid cylinder, without any perforation, is the best instrument for exploring the action of the heart; but as this form is not so good for examining the voice and respiration, the perforated cylinder is commonly used for all purposes."

Having developed a suitable stethoscope, Laennec undertook his epoch-making observations.

"I commenced immediately at the hospital Necker a course of observations, which have resulted in the discovery of new signs, sure, for the most part obvious, easy to be possessed of, and suitable to render the diagnosis of almost all diseases of the lungs, the pleuras, and the heart, more certain, and perhaps more circumstantial, than even the surgical diagnostic signs established by the aid of the probe or the finger." (Traité de l'auscultation médiate, tome 1, p. 8.)

Piorry's Improvement.—Piorry (1828) modified the Laennec stethoscope by reducing it to the thickness of a finger. He constructed an ear piece which overcame the difficulties experienced by some physicians

in obtaining a proper seal to the ear. Piorry's modification included a trumpet-shaped chest piece. Essentially, his instrument is the modern monaural stethoscope.

The Binaural Stethoscope.—The exact date of the first binaural stethoscope is unknown; several people have been credited with this invention. In 1907, C. T. Williams, in a paper in the *British Medical Journal*, described a stethoscope invented by his father (C. J. B. Williams) in 1829, and stated that it was probably the first binaural stethoscope ever made. In any event, Dr. C. J. B. Williams did describe a binaural stethoscope about 1843, and Dr. Arthur Leared demonstrated a binaural instrument in 1851. In Dr. Williams' early instrument, the tubes connecting the chest piece with the ear pieces were made of lead, and it was not until Dr. George P. Cammann first developed the binaural stethoscope with the flexible tubes that the true precursor of the present binaural stethoscope appeared. Cammann described his instrument in 1855. The chest pieces of all of these stethoscopes had a funnel or trumpet shape, with the acoustic characteristics of the so-called bell stethoscope. It was not until the invention of the diaphragm chest piece, with a rigid covering over the end of the collecting part, that a significant modification of the older bell type was developed. Dr. Marsh, of Cincinnati, in 1851, patented a stethoscope with a flexible membrane stretched over the end, but the phonendoscope devised by Bianchi, in 1894, and described also by Bazzi, in 1895, was the first instrument to have a rigid diaphragm. It was further modified by Baruch, of New York, in 1896. The chest piece of this type, which is in common use in this country at the present time, is the one patented by Dr. R. C. M. Bowles, of Brookline, Massachusetts, June 25, 1901. The importance of all these instruments rests in the fact that the rigid diaphragm acts to attenuate the low-pitched sounds and thereby accentuates the faint, high-pitched, blowing murmurs which are heard most characteristically in slight aortic regurgitation. Connor, in 1907, pointed out that this type of murmur can at times be heard best by applying the naked ear to the chest wall.

It would seem to be agreed at the present time that the bell chest piece and the diaphragm chest piece can satisfactorily cover the acoustic ranges necessary for clinical examination of the heart. However, the development of modern electric amplifying systems, with their freedom from background noise, has opened a new field for the graphic registration of heart sounds and murmurs, as well as respiratory sounds, which may well modify our clinical opinion in the future.

III. Characteristics of the Human Ear in Auscultation

In auscultation, the human ear may be considered as the recording mechanism of an acoustic system. H. D. Arnold, in his introduction to

Harvey Fletcher's "Speech and Hearing,"[2] which is considered one of the best treatises on the subject from a physical standpoint, states:

"Our ears are only machines to translate air waves into a form suited to stimulate the auditory nerve; and as machines we may measure and describe them in the same terms that apply to devices we ourselves construct. We may compare them as to performance, and may accommodate our devices to their requirements. But, to understand the mechanism of the ear is by no means to understand the act of hearing, for we have not heard until the brain has perceived the message sent by the auditory nerve. We cannot explain in precise mechanical terms how this is done, nor indeed have we any clear comprehension of the process at present. Some important factors relating to the process of hearing we can, however, determine by measuring the least changes in sound which can be detected under a variety of conditions of pitch, loudness, and accompanying noise. Thus we may obtain a quantitative means of comparing individuals in this respect, and establish a standard of average hearing."

Threshold of Hearing and Feeling vs. Frequency.—Pure tones of different periods of oscillation or frequency, but of similar intensity, affect the human hearing mechanism to different degrees. Investigations which ascertained the actual intensity of sound at the threshold of audibility were made by Fletcher,[2] Toepler and Boltzmann,[3] Rayleigh,[4] Wead,[5] Wein,[6] Abraham,[8] and Kranz.[9] Wegel[10] investigated the intensity of sound at the threshold of feeling.

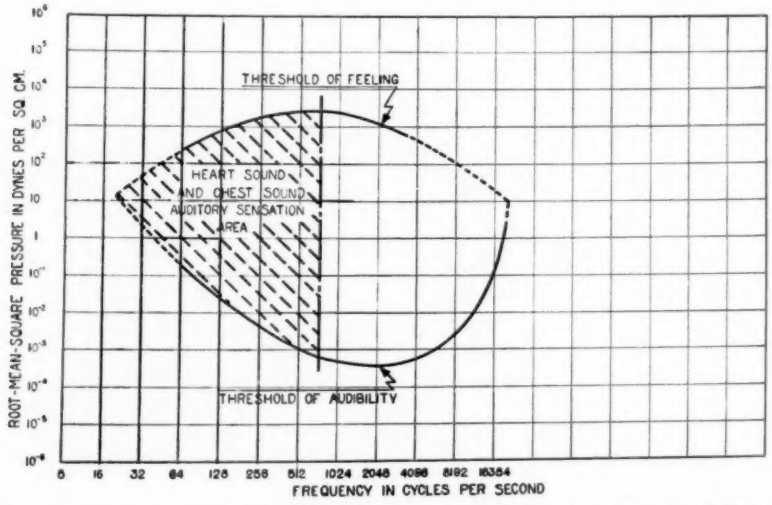

Fig. 1.—Average normal threshold curves of audibility and feeling (modified from Fletcher).

Fig. 1 shows the curves representing average thresholds of feeling and audibility which were obtained on a large number of persons by Fletcher. The threshold of audibility curve represents the pressure variations in dynes per square centimeter, or bars, which are necessary to produce a sound of minimum audibility between the frequency limits of approximately 20 and 20,000 cycles per second. The threshold of

feeling curve represents the pressure variations beyond which a sensation of pain is created. The area enclosed by the upper and lower curves represents the auditory sensation area. The threshold curves of any one person would not be so smooth (peaks and valleys are superimposed), for there are hardly two sets of ears that are identical. The broken portions of the feeling and audibility curves are obtained by extrapolation, which is made necessary by the fact that the phenomena are not clearly defined in these regions. The shaded area represents the auscultation region with which we are concerned.

The Decibel.—The human hearing mechanism is capable of functioning over an enormous range of sound pressure or sound energy variations. To express this property of hearing in quantitative terms of pressure or energy would require the use of numbers of such magnitude that their arithmetical manipulation and visualization would be impractical. The communications engineer was confronted with this problem and overcame it by employing the decibel system of measurement.

The bel is essentially a logarithmic unit, or the common logarithm of the ratio of two values of sound power, and the decibel, as the term indicates, is one-tenth of the bel. An approximate characteristic of hearing is that equal variations of sound intensity along a logarithmic or decibel scale approximate similarly equal variations of loudness as they are perceived by a normal human ear. Also, a one-decibel variation in power level of sound is approximately the minimum change that may be detected by the average human being.

In the decibel scale relative sound intensities are always expressed in terms of differences in decibels, never as a ratio of decibels or a percentage change in decibels. From the definition of the decibel, one decibel merely represents a sound pressure ratio equal to the twentieth root of 10, or a ratio of 1.12 to 1. That is, two sounds which differ by one decibel have their pressures in the ratio of 1.12 to 1. Similarly two sounds which differ by 20 decibels have their pressures in the ratio of the twentieth power of 1.12, or 10 to 1. If a sound pressure level is doubled, the change in decibels is twenty times the logarithm of 2, which is 6 decibels. Thus, doubling a given sound pressure always increases by 6 decibels the original level, no matter what the value of the original level was.

In order to obtain a better conception of the relative values in a decibel scale, let us consider Table I. In the application of the decibel scale to the measurement of the intensity of sound, a reference point, such as the threshold of hearing, is commonly employed. Table I lists the average decibel equivalents above the hearing threshold of various sources of noise. The instantaneous values may vary plus or minus 10 decibels from the values given. The first three columns were obtained in a survey by the New York City Noise Abatement Commission. The fourth column was added by us to illustrate the tremendous range in intensity that exists betwen the threshold of hearing and the threshold of pain. Column four is merely an expression of column three, converted into equivalent intensity ratios with the threshold of hearing as the reference value.

TABLE I

SOURCE OR DESCRIPTION OF NOISE	DISTANCE FROM SOURCE (FEET)	NOISE LEVEL (DECIBELS)	NOISE LEVEL (AMPLITUDE RATIOS)
Threshold of pain		130	3,162,000
Hammer blows on steel plate (almost painful)	2	114	501,200
Riveter	35	97	70,790
Elevated train	15-20	90	31,620
Average motor truck	15-20	75	5,620
Busy street traffic	15-75	68	2,512
Average automobile	15-50	66	1,995
Ordinary conversation	3	65	1,778
Rather quiet street (residential)	15-300	58	794.0
Quiet automobile	15-50	50	316.2
Average office		47	223.9
Noisy residence		45	177.8
Very quiet radio in home		40	100.0
Average residence		32	39.8
Average whisper	4	20	10.0
Rustle of leaves in gentle breeze		10	3.1
Threshold of hearing		0	1.0

Minimum Perceptible Changes in Intensity and Frequency.—A law in psychology, known as the Weber-Fechner law, states that the stimulus increase required to produce a discernible sensation increase maintains a ratio which is constant to the total stimulus. Another way of stating the Weber-Fechner law is that the magnitude of the produced sensation is proportional to the logarithm of the stimulus. When the Weber-Fechner law is applied to hearing, it may be stated that a barely perceptible increase in sound intensity, as detected by the human ear, should exhibit itself as a constant which is independent of the sound intensity.

Knudsen[11] is to be credited with the first accurate determination of the minimum perceptible variations in intensity between the frequency limits of 100 and 4,000 cycles per second. Fletcher,[2] by a modification of the testing procedure, extended the frequency band between the limits of 35 and 10,000 cycles per second. Knudsen and Fletcher observed some variation in the Weber-Fechner law, as applied to hearing, in that the average ear was most sensitive to variations in intensity at sensation levels above 50 decibels, and particularly did this hold true in the middle of the frequency band. An intensity variation of 5 to 10 per cent was detectable for sound levels above 50 decibels at approximately 2,000 cycles per second. At the lower sound levels, and more so at the extreme ends of the frequency band, a decidedly greater percentage variation in intensity was required to produce a perceptible change. As a comparative illustration, it was found that a 70 per cent variation in intensity was necessary at 2,000 cycles per second in order to produce the minimum perceptible change when the sound level was down to 10 decibels.

In auscultation, this characteristic of human hearing is an extremely important factor, in that the intensity level is most often 10 decibels, or less, in addition to the fact that the frequency band which is involved is at the lower extreme. As an illustration, at a frequency of 60 cycles per second a variation of 20 per cent is just perceptible when the sound level is 50 decibels, and a variation of as much as 200 to 300 per cent is necessary at the intensity level of 10 decibels.

The human ear is a far better detector of changes in frequency than it is of changes in intensity. Shower and Biddulph,[12] as well as Knudsen, have made extensive investigations in order to ascertain the minimum perceptible frequency changes at the various sensation levels. They found that a higher sensation level of sound required less of a frequency change for minimum perceptibility. Also, the ear was found to be less sensitive to frequency variations as the lower end of the frequency band was approached. Between 500 and 4,000 cycles per second, the minimum perceptible fractional difference in frequency is 0.3 per cent at a sensation level of 40 decibels. At 64 cycles per second the minimum perceptible fractional difference in frequency is of the order of 1.1 per cent; at 128 cycles per second it is 0.6 per cent; and at 256 cycles per second it is 0.4 per cent.

Minimum Tonal Perception Time.—Another important measurement pertaining to auscultation is the minimum amount of time required for a tone to excite the hearing mechanism. Stewart's[13] uncertainty principle states that the ability of the ear to recognize a tone of brief duration decreases as the duration decreases. Stewart expressed his principle mathematically by saying that the product of the duration of the tone by the number of cycles of uncertainty in ascertaining the pitch of the signal is equal to unity. It naturally follows from this expression that, in the lower frequency band, such as is encountered in auscultation, the frequency may be varied rapidly over a considerable portion of an octave without detection by the ear. This phenomenon may be illustrated if a vibrato with a frequency of 60 cycles per second is varied by two semitones at a rate of seven times per second; the variation cannot be detected by the ear. Table II (after Fletcher) presents some probable values as regards tonal perception.

TABLE II

FREQUENCY	WEAK TONES		MEDIUM TONES	
	TIME (SECONDS)	CYCLES	TIME (SECONDS)	CYCLES
128	0.0946	12.1		
256			0.06908	17.6
384	0.0627	24.08	0.0445	17.1
512	0.0579	29.64	0.04274	21.8

Masking Effects.—Everyone has noted that in a rather noisy location he unconsciously increases the intensity of his voice in order to make

himself heard. The reduction in the ability of the ear to detect certain sounds in the presence of other sounds is technically known as masking. Mayer,[14] in 1876, was one of the first to show experimentally that low-pitched sounds had a masking effect which differed from that produced by high-pitched sounds. Mayer claimed that a low-pitched tone was capable of completely masking a tone of higher pitch, but that a higher pitched tone was incapable of completely masking the lower pitched one. The equipment used by Mayer in his experiments was rather crude, so that the accuracy of his work was limited. The Bell Telephone engineers continued the study and obtained accurate data which were published in Fletcher's "Speech and Hearing."

Experimenters in the Bell Telephone Laboratories found that Mayer's conclusion on masking holds true only under certain conditions. A low-pitched tone will not mask to any degree a high-pitched tone far removed in frequency unless the low-pitched tone is of a very considerable intensity; a higher pitched tone may easily mask a lower pitched tone if the frequencies are closely spaced.

In a complex tone, as, for example, one consisting of three frequencies, such as 300, 400, and 2,000 cycles per second, with respective sensation levels of 50, 10, and 10 decibels, the experimenters found that the average human ear could detect only the 400- and 2,000-cycle tones. When the intensity of each component of the complex sound was increased by 30 decibels, so that the resulting three tone levels were 80, 40, and 40 decibels, respectively, it was noted that the 300- and 400-cycle tones were the only ones audible. Under these conditions, when the 300-cycle tone was attenuated by only 8 decibels, it vanished completely. From this example it follows that the sensation produced on the ear by a complex sound is decidedly different in character, as well as in intensity, when the level is decreased or increased, even though no distortion is introduced. *This peculiar characteristic of hearing follows the general rule that, as a complex sound becomes more intense, the low-pitched tones will become more prominent because of the fact that the higher pitched tones are masked.*

When a radio receiver is tuned to a station which is broadcasting a symphony, this masking effect will readily become apparent if one listens first at high volume, and then turns the volume control to a much lower level. At the high volume setting the bass notes come through with excellent fidelity, but at the low volume setting a decided attenuation in the bass will immediately become apparent.

In auscultation this masking effect is even more noticeable because of the proximity of the frequency components which are involved. It follows that obesity should have an attenuating effect and produce a decided change in the quality of the heart sounds. Furthermore, some of the important components of the heart sounds might be completely masked by obesity.

Another type of masking effect is introduced, especially in auscultation, when a sound of comparatively large volume immediately precedes a sound of considerably less volume, as, for example, when a first or second heart sound precedes a murmur of low intensity. The first or second sound of comparatively great intensity has a tendency temporarily to fatigue the ear, thereby masking the low intensity murmur. The diaphragm type of stethoscope chest piece partially overcomes the masking effect by selective attenuation, the principle of which will be discussed later.

Frequency Response Characteristic.—Because of the fact that the human ear does not respond equally to different pitches, tones of the same intensity but of different frequencies produce different sensations of loudness. Also, if the intensities of the tones of different pitch but of equal loudness are increased by an equal amount, an unequal sensation of loudness is produced. Kingsbury[15] and, more recently, Fletcher and Munson[16] obtained quantitative measurements on the determination of equal loudness curves for average subjects.

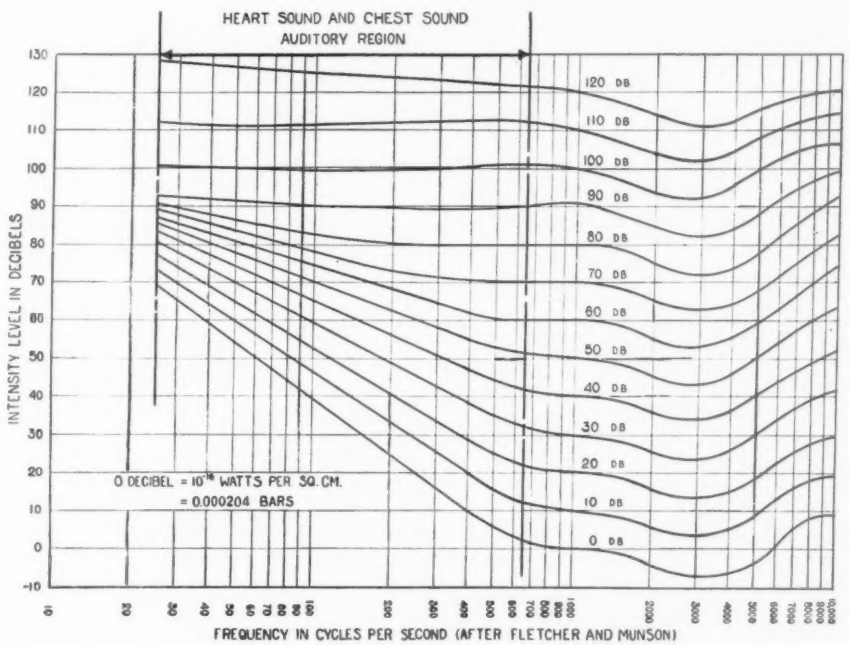

Fig. 2.—Relative sensitivity curves of the average normal human hearing mechanism.

Fig. 2 shows the curves obtained by Fletcher and Munson. The abscissas of the graph, which is a logarithmic scale, represent the frequency band in cycles per second that is covered by the average human ear, and the ordinates are the intensity levels, applied to the ear, in decibels. The family of curves was obtained for equal steps of increase in intensity (indicated on each curve in decibels) above zero decibels,

which represent the threshold level and are equivalent to $\frac{1}{10,000,000,000,000,000}$ or 10^{-16} watts per square centimeter of sound excitation on the ear, or 0.000204 bars.

From the family of curves, it may be seen that the maximum sensitivity of the average ear is in the vicinity of 3,000 cycles per second. In the frequency band above 1,000 cycles per second, a given increase in the intensity level results in approximately the same increase in loudness, whereas in the very high, and, more so, in the low frequency regions, lesser increases in intensity produce a greater sensation of loudness. Resolving this statement into actual values from Fig. 2, at a frequency of 600 cycles per second an increase in intensity of 76 decibels is necessary to create a loudness level of 80 decibels above the threshold level of zero decibels, whereas, at 100 cycles per second, an increase of about 43 decibels is necessary to produce an equal increase; at 8,000 cycles per second, 80 decibels are required, and, at 1,000 cycles per second, 80 decibels. It is very important that this nonlinear characteristic of the ear be taken into consideration when designing a stethoscope, for a modification of the frequency response characteristic of the stethoscope will seem to alter the quality of the heart and chest sounds.

IV. Description of Heart Sounds, Murmurs, and Noncardiac Chest Sounds From a Physical Point of View

Transmissional Characteristics.—Acoustics, as related to auscultation, deals with the vibrations or disturbances set up in the chest and their transmission through the component sections of the chest, as well as through the stethoscope, and with the resulting effects on the observer's ear. Sound, which is the result of vibration of a medium, is capable of traveling through a solid, liquid, or gas as a compressional wave, unless the medium is entirely inelastic. In other words, the ability of sound to travel through matter depends upon the elasticity, viscosity, and density of the medium. If the effects of viscosity are small, as is the case with water, air, metal, and bone, the sound energy may be transmitted with little loss. In some media, such as soft rubber or fatty breast tissue, the sound waves are almost immediately suppressed.

In a homogeneous substance a sound wave will propagate itself at a velocity whose magnitude depends on the physical properties of the medium, and the attenuation of the wave will be governed by the viscosity and the spreading of the sound energy over a larger surface as the wave progresses. When sound energy travels from one medium into another of different physical properties, or when there is no longer homogeneity of medium, transmission losses in the form of refraction and reflection take place. Some media are capable of transmitting low frequency vibrations with less attenuation than high frequencies, and vice versa; this results in a distortion of the sound.

There are many paths along which heart and chest sounds travel in the human body in order to reach the surface. A large percentage of the sound energy never reaches the surface because of viscosity, elasticity, density, spreading, reflection, and refraction losses. Naturally, the intensity of the sound is maximum over the portion of the chest where

the sound pursues a path of minimum attenuation, and this location the physician normally selects with his stethoscope. Auscultatory experience has definitely shown that murmurs produced by lesions of the four valves of the heart are heard with maximum intensity in certain areas of the chest, although there may be other sections of the chest surface which are closer to the valves under observation.

Classification of Sounds.—Trendelenburg,[17] Williams and Dodge,[18] Pierach,[20] Landes,[21] and Cabot and Dodge[22] have attempted to ascertain what frequency band is involved in auscultation. Cabot and Dodge[22] came to the following conclusions:

"Practically all the sounds of interest in auscultation are made up of frequencies below 1,000 cycles per second. In general, the upper and lower frequency limits for the pathologic sounds in any particular case are not sharply defined.

"Presystolic murmurs as a class are characterized by a greater predominance of low frequencies than other murmurs, and are almost invariably termed low pitched.

"The frequency bands of importance in systolic and diastolic murmurs are broadly the same. In each group, extremely low-pitched and high-pitched cases are found, but there appears to be nothing in the quality of the sounds that is characteristic of murmurs occurring in systole or diastole. In our limited studies, we have not been able to associate a particular frequency band with murmurs produced by a lesion of a given kind.

"Lung sounds, as a class, have a proportionately smaller amount of low frequency components than heart sounds. The descriptive words 'coarse' and 'fine,' as applied to râles, are usually associated with conspicuous 'low' and 'high' frequency components. Breath sounds are, broadly, higher pitched than most heart sounds, as evidenced by the scarcity of components below 240 cycles per second."

Williams and Dodge[18] observed the distribution of energy in the normal heart sounds as given in Table III.

TABLE III

FREQUENCY (CYCLES PER SECOND)	ENERGY (PER CENT)
50-60	56
60-70	27
70-80	10
80-90	4
90-100	2
100-110	1

Above 110 cycles per second, the energy components of the normal heart sounds were found to be negligible. Williams and Dodge further observed that low-pitched heart murmurs are composed of frequency components below 400 cycles per second and that high-pitched murmurs range from 120 to 660 cycles per second. Systolic and diastolic murmurs are composed principally of energy components between 120 and 660 cycles per second, occasionally ascending to 1,000 cycles per second. Cabot and Dodge[22] noted that the frequency of presystolic murmurs lies,

for the most part, below 140 cycles per second, but they may contain components up to 400 cycles per second. They stated that the pericardial rub is composed of frequencies between 140 and 660 cycles per second, that râles lie between 120 and 1,000 cycles per second, amphoric breathing between 240 and 660 cycles per second, and bronchial breathing between 240 and 1,000 cycles per second.

In our observations on the frequency components of the various heart sounds and murmurs, we have not encountered any of noticeable value above 650 cycles per second. We have, however, encountered frequency components of murmurs and heart sounds that were well below the range of the human ear (obtained by recording the sounds graphically). Just where the lower limit of the sounds actually occurred was rather difficult to observe accurately, for they were intermingled with the low frequency chest wall motions. The lowest frequency limit of heart sound and murmur components is in the vicinity of 5 to 10 cycles per second, although sounds with a frequency of less than 30 to 40 cycles per second cannot be heard if the intensity is low.

V. The Acoustic Stethoscope

General Classification.—The acoustic stethoscopes may be classified as monaural, binaural, and differential. The monaural stethoscope described earlier in this paper was invented by Laennec and later improved by Piorry. It is still in use on the continent of Europe, but the binaural stethoscope is more commonly used. There are many kinds of binaural stethoscopes, but the only two really important types are those with open and diaphragm chest pieces.

The differential stethoscopes are primarily instruments for localizing and comparing sounds. Their operation depends on the well-known characteristic of hearing termed "auditory localization," or the ability to ascertain the direction of a sound source by means of phase differences. Phase difference depends on when the sound reaches each ear, and, in turn, registers on the brain. In addition, there is a stereophonic effect which gives the sound characteristic depth and extensiveness.

Alison,[88] in 1859, described a differential stethoscope which consisted of two monaural stethoscopes of the flexible-tube type. Muralt,[86] in 1910, devised a differential stethoscope which allowed him to listen simultaneously to two lung areas. Muralt's stethoscope incorporated two similar chest pieces, so arranged that two tubes of equal length connected each chest piece with each ear; four tubes were used in all. In 1934, Fröschels[83] suggested using a differential stethoscope to study the sounds produced by the vocal cords in order to detect laryngeal paralysis. Fröschels' stethoscope was a modification of Muralt's, in that it consisted of two similar chest pieces and two tubes passing through an X which allowed an ipso- or contralateral course of sound to each ear. Hawthorne,[89] in 1935, described a differential (double) stethoscope which was similar to Alison's. In 1936, Nicolai[84] described what he termed a "Stereostethoscope," to detect differences in sounds originating in the two mandibular joints. The Nicolai "Stereostethoscope" was identical with the one described by Alison. Nicolai and Hantschmann[87] found the Nicolai "Stereostethoscope" useful in the study of pulmonary and cardiac disease.

Kerr, Althausen, Bassett, and Goldman,[85] of the University of California Medical School, described another instrument which they called the "symballophone" and classified it as a "modified stethoscope for the lateralization and comparison of sounds." The "symballophone" is similar to Muralt's stethoscope, except that a longer connecting tube from each chest piece to the opposite ear piece is employed in place of the direct tube to the ear piece on each side.

Many variations and styles of bells have been manufactured and marketed with wide claims for their performance, but rarely has a theoretical or scientific explanation of the acoustics of the bell been attempted. Similarly, the acoustics of the stethoscope structure between the bell and the ear has been ignored. With due credit to the contribution of medical men to the development of the stethoscope, the fact cannot be overlooked that this commonly used instrument is lacking in certain elements of scientific design.

Principle of Operation.—The stethoscope is a device which provides a closed acoustic system for conducting the sounds that originate in the body of the patient to the observer's ear. The sound transmission medium is a closed column of air in the case of the binaural stethoscope, which employs rubber tubing as a link between the bell, or sound accumulator, and the ear pieces. In the rigid monaural stethoscope, the sound travels by both bone conduction and air column transmission. The bone conduction is dependent upon contact with the ear as well as with the material of which the stethoscope is made, whereas transmission by air column depends upon the pressure changes that take place.

The Open Bell.—In medical literature the limited references to attempts to analyze the acoustics of the bell portion of the stethoscope show that the unit is regarded merely as an accumulator or collector of sounds. The general trend of thought has been that, if the sound as it accumulates on a patient's chest could be gathered from the largest practical area, there would be a proportional increase in intensity when it is transmitted to the ear (Barss, Eade, and Fitzgerald,[90] Tobler[91]). According to this reasoning, the larger the diameter of the contact area of the bell consistent with good seating, the greater the heart sound intensity. Limited clinical trial seemed to substantiate this supposition. Various types of bells whose construction was based upon these assumptions have been marketed. Other designers went a step further by giving the internal portion of the bell various geometrical shapes in order to aid the accumulating properties.

It is our opinion that the open bell should not be considered primarily as an accumulator. When it is held in the open, the open bell exhibits acoustic characteristics that are entirely different from those which it exhibits when it is applied to the chest. *When an open bell is applied to the chest, the skin bounded by the lip of the bell forms a diaphragm, and the fleshy portion under the skin acts as a damping medium.* In other words, the condition is somewhat similar to that encountered in a microphone or telephone receiver.

The physical principles governing the operation of a damped diaphragm are well known and may be found in textbooks on telephony. Any diaphragm has a natural period of vibration, or resonance point, which is dependent upon its inherent inertia, elasticity, diameter, and tautness. The maximum sensitivity of the diaphragm to external excitation occurs at the point of resonance. In other words, if a diaphragm (made of any material) is excited by sound or vibrational energy whose pitch or frequency is varied from zero to infinity while the amplitude is kept constant, the diaphragm will oscillate in unison with the exciting medium and reach a maximum oscillation amplitude at its point of resonance. The following are some of the general rules governing the action of diaphragms:

A. The more taut a diaphragm is drawn, the higher does its natural period of oscillation become.

B. The larger the diameter of the diaphragm, the lower will be its natural period.

C. Damping serves the purpose of suppressing the maximum vibrations of the diaphragm that occur at the resonance point.

D. When the natural period of a diaphragm is increased, the upper frequency range to which the diaphragm is capable of responding is increased. This effect cannot be attained, however, without lowering the sensitivity of the diaphragm throughout its entire frequency range. Thus, a diaphragm with a higher natural period is less sensitive to the lower frequencies or pitches than a diaphragm of lower natural period.

E. Diaphragms have harmonic resonance points which occur at multiple frequencies of the fundamental. In auscultation, we are concerned only with the fundamental.

F. At the frequency of mechanical resonance of the diaphragm the effects of the inertia and elasticity of the diaphragm balance each other, and its velocity is in phase with the impelling force.

If we regard the open bell of the stethoscope as a device for producing a diaphragm effect when it is applied to a patient's chest and keep in mind the physical characteristics of diaphragms, many clinical observations become explainable. It is a commonly observed fact in auscultation that, for a bell of a given size, the greater the pressure with which the bell is applied to the patient's chest, the less the apparent intensity of the first and second heart sounds and the higher the pitch. This phenomenon is readily explained by the fact that, with greater pressure, the skin bounded by the lip of the bell is drawn more taut; this produces a diaphragm with a higher natural period than when the pressure is light. As has been said, a diaphragm with a high natural period extends the upper portion of the frequency band, but, at the same time, attenuates the over-all level except at the point of resonance. It is to be expected that a diaphragm with a high natural period would attenuate the lower frequency components of the heart sounds and tend to bring out the higher frequency components, thereby altering the pitch and reducing the intensity of the first and second heart sounds, in which the low frequencies predominate.

It is noted normally that the smaller the diameter of the opening of the bell, the higher the pitch of the sounds. This characteristic is ex-

plained by the fact that, with a small bell, the diaphragm dimensions are small, and this tends to give it a higher natural period. Also, in order to obtain a reasonably good seal with the skin, sufficient pressure is exerted to stretch the skin enclosed by the lip of the bell, thereby additionally raising the natural period. With a bell of larger diameter, the tendency to stretch the skin is proportionally reduced; this makes a stethoscope with a bell of large diameter insensitive to pressure variations as regards pitch, whereas a stethoscope with a small bell is very susceptible.

The pressure-pitch characteristic of stethoscopes has a useful application in auscultation when a bell of normal size (about one inch in diameter) is employed. By varying the pressure on the patient's chest, the physician creates a variable filtering action upon the heart sounds that are transmitted by the closed system. In order to clarify the principle, let us consider a pathologic condition in which there are normally loud first and second heart sounds, a loud systolic murmur of fairly low pitch, and a very low intensity, hardly audible, high-pitched diastolic murmur. Because of the comparatively great intensity of the sounds other than the diastolic murmur, the latter is masked (as explained under *Masking Effects*). When the pressure with which the stethoscope is applied is increased, the natural period of the diaphragm formed by the skin is raised, thereby attenuating the lower pitched components, of which the first and second heart sounds, as well as the systolic murmur, are composed, and allowing the higher pitched diastolic murmur to stand out better as a result of the decreased masking effect. When the pressure with the stethoscope is decreased, low-pitched murmurs, third heart sounds, and gallop rhythms may be brought out more distinctly.

Another very important consideration in regard to the open bell is the effect of its internal dimensions and shape. As has been previously mentioned, various geometrical shapes and forms were devised to improve the accumulating properties of the open bell, with a resulting loss of accumulation in most cases. The fallacy in making bells of certain geometrical shapes becomes obvious if we consider the stethoscope from the point of view that the pressure variations at the ear which are produced by the skin diaphragm in the stethoscope proper are inversely proportional to the volume of the bell. Thus, an infinitely small volume produces a maximum variation in pressure, which, in turn, manifests itself as a sound of maximum intensity. The only important consideration in designing a bell, aside from keeping the internal volume at a minimum, is to have it so shaped that, in the case of an obese patient, the bell will not fill with flesh to such an extent as to decrease the diameter of the diaphragm and, therefore, its effect.

At this point it is logical to consider the relationship of bell resonance to dimensions. A stethoscope bell may be placed in the same class as the Helmholz resonators, an analysis of which may be found in almost any text on acoustics. Although it is somewhat beyond the scope of this paper to go into the acoustics of

Helmholz resonators as applied to stethoscope bells, it is of interest to note that a bell with an internal lip diameter of 5.8 cm. and an internal body depth of 0.9 cm., with a 0.6 cm. hole, 1 cm. long, through the threaded portion (which attaches to the stethoscope proper), has a theoretical resonance frequency of approximately 500 cycles per second. Because of the fact that so large a bell has a resonance frequency which lies at about the upper limits of heart and chest sound frequencies, and bells of smaller size exhibit proportionally higher resonance frequencies, *it is impossible to obtain any really effective filtering by bell shape modifications other than those previously mentioned.*

The Diaphragm Chest Piece.—The diaphragm chest piece (Bowles type), which is commonly employed in auscultation, is especially useful in detecting faint, high-pitched sounds, such as the barely audible, high-pitched, diastolic murmur of aortic insufficiency and the high-pitched "bronchial" respiratory sounds. Essentially, the principle of its operation is similar to that of the open bell when it is applied to the patient's chest, except that additional attenuation of the lower pitched heart and chest sound components is obtainable. Of course, the smaller the diameter of the open bell, and the greater the pressure with which the bell is applied, the greater the low frequency attenuation, but, to obtain an equivalent degree of attenuation with the open bell, the pressure would be great enough to hurt the patient. By interposing a diaphragm of bakelite, or any other plastic whose natural period is in the desired range, the necessary attenuation is obtainable with light pressure on the patient's chest. In other words, the plastic diaphragm becomes a substitute for the skin diaphragm, and the flesh of the chest acts as a damping medium.

All of the general principles which are applicable to the skin diaphragm apply equally well to the plastic diaphragm. It is well to mention at this time that the plastic diaphragm is referred to because it is the one most generally employed. The theory of operation of a metal diaphragm is identical, but a foreign, metallic ring may be superimposed upon the sounds.

As is the case with the open bell, for maximum efficiency the volume of air in the diaphragm chest piece should be as small as possible. The internal volume of a diaphragm bell may be considerably less than that of an open bell of similar diameter, for the stiff diaphragm prevents the tissue of the patient's chest from entering and decreasing the effective diameter.

Open Bell vs. Diaphragm Bell.—An attempt was made to verify experimentally the theory of the stethoscope bell which has been discussed. A frequency characteristic run was made with the four bells shown in Fig. 3. Fig. 4 is a schematic sketch of the apparatus employed in the experiment.

The posterior portion of the patient's chest was allowed to rest against the loud-speaker, with its associated baffle. Throughout the entire test the patient was kept in one position relative to the loud-speaker. The four bells of Fig. 3 (the bells may be screwed into the specially designed microphone, the characteristics of which will be discussed later) were placed one at a time on an area marked on the

Fig. 3.—Chest pieces employed in verification of stethoscope bell theory.

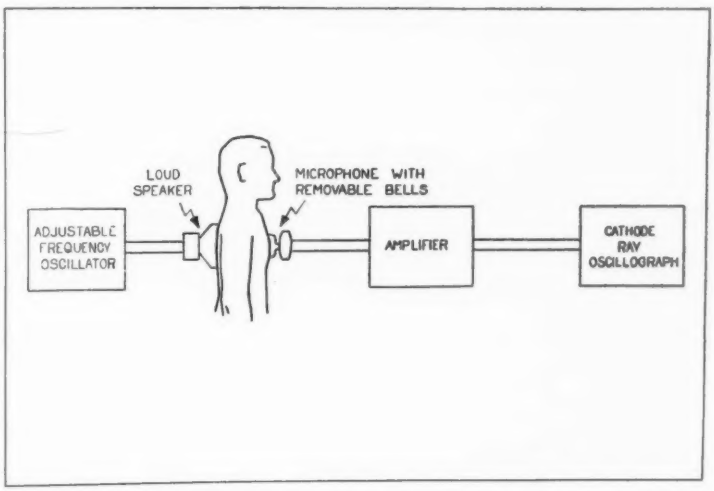

Fig. 4.—Schematic diagram of apparatus employed in obtaining Fig. 5.

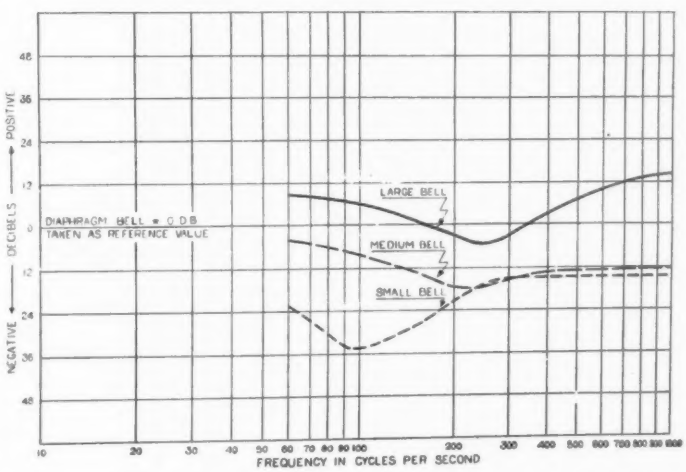

Fig. 5.—Resultant curves of the three open chest pieces, as compared with the diaphragm chest piece. The higher the decibel reading, the higher the relative efficiency of the chest piece.

front of the patient's chest. In other words, the adjustable frequency sound energy was fed into the patient's chest and picked up by the microphone and its associated bells; it was then amplified and fed into a cathode-ray oscillograph, used as a recorder. Normal pressure on the patient's chest was used with all four bells.

A frequency response curve was recorded for each bell. The curves thus obtained had practically no meaning, for they included the characteristics of the loud-speaker and the transmissional media of the chest. However, subtraction of one response curve from the other canceled the loud speaker and chest transmissional characteristics, thus allowing direct comparison of the respective bells. For our purpose, a comparison of the diaphragm bell with the open bells (using the diaphragm bell as a reference value) supplied useful information. In Fig. 5 the resultant curves of the three open bells and the diaphragm bell are compared; the reference value was zero decibels.

The dimensions of the four bells were as follows: The lip diameter of the large bell was 2.0 inches, of the medium bell, 1.5 inches, and of the small bell, 1.0 inch. The diaphragm bell was a typical "Bowles" chest piece, with a diaphragm 0.015 inch thick and a freely working diameter of 1⅜ inches. The internal volume of the large bell was 12.7 c.c., of the medium bell, 6.2 c.c., of the small bell, 2.3 c.c., and of the diaphragm bell, 2.5 c.c.

The following are some of the more important general conclusions that may safely be drawn from the graph of Fig. 5:

A. The efficiency of the three open bells improves, with respect to that of the diaphragm bell, as the frequency is decreased.

B. The larger the diameter of the open bell, the more efficient is the bell at the lower frequencies.

C. The large bell exhibits a resonance effect in the upper auscultatory region that is characteristic of a Helmholz resonator of such dimensions. The resonance points of the smaller bells are above the auscultatory range and therefore are not shown in the graph.

It has been previously mentioned that a diaphragm bell is useful in bringing out certain high-pitched murmurs because it suppresses the lower frequency heart sound components that tend to mask the low intensity murmur of high pitch. The commonly used diaphragm bell, such as the Bowles of Fig. 3, employs a bakelite diaphragm 0.015 inch thick, with a freely working diameter of 1⅜ inches. This diaphragm was selected because clinical trial showed that it suppresses the lower pitches sufficiently well to bring out the higher pitched murmurs that are normally masked. Thicker diaphragms, or diaphragms with a higher natural period are not normally used because they produce such a pronounced over-all attenuation of the heart sounds that they are more of a disadvantage than an advantage. If the efficiency of the acoustic stethoscope could be increased enough to overcome the general reduction of sound intensity which occurs when thicker diaphragms are employed, the hard-to-hear, high-pitched diastolic murmurs might be more easily detected. This proved to be true when an amplifying stethoscope was employed; a diaphragm chest piece with a higher natural

period and an adjustable volume control was used. This test will be discussed in greater detail later.

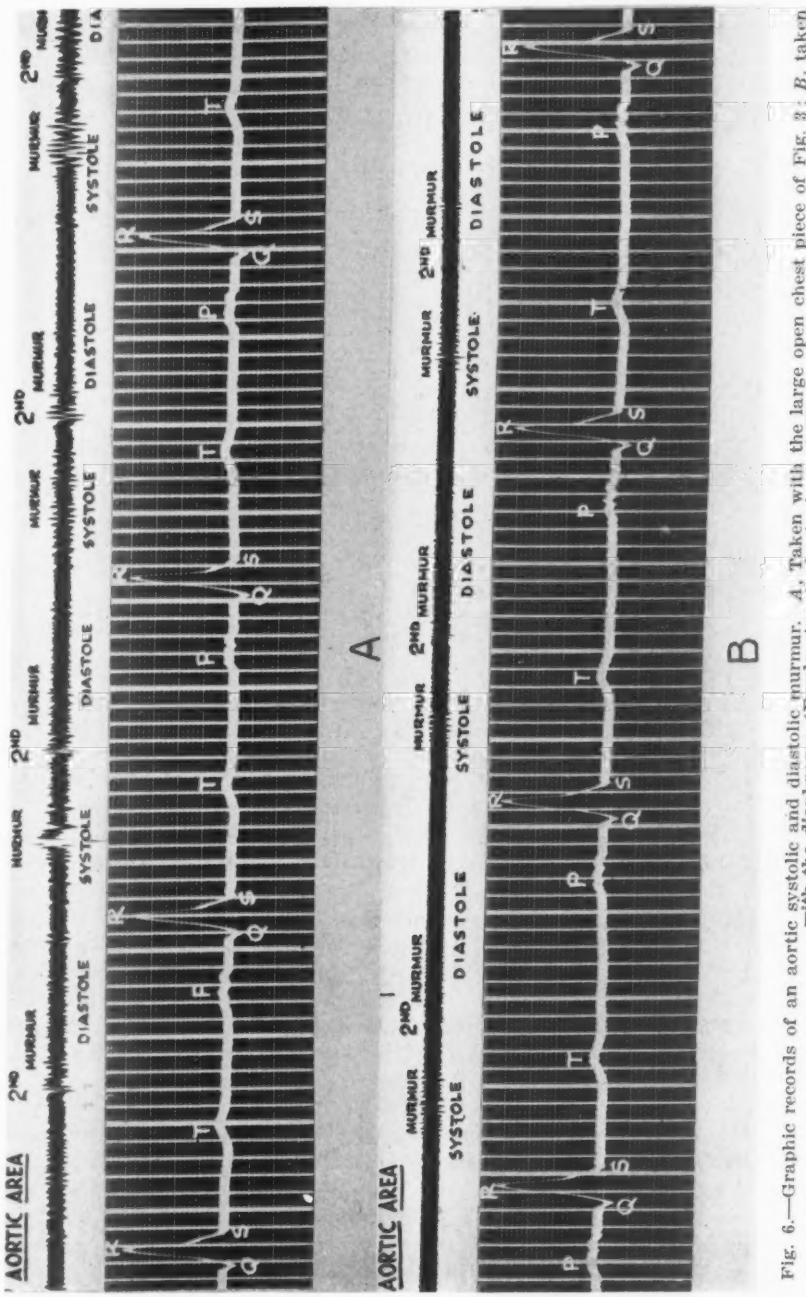

Fig. 6.—Graphic records of an aortic systolic and diastolic murmur. *A*, Taken with the large open chest piece of Fig. 3; *B*, taken with the diaphragm (Bowles type) chest piece of Fig. 3.

Fig. 6 shows two records which were obtained on a patient with an aortic systolic and diastolic murmur. Fig. 6*A* was made with the large

open bell of Fig. 3, and Fig. 6*B* with the diaphragm bell of Fig. 3 (bakelite diaphragm, 0.015 inch thick). The filtering action, or attenuation, of the low frequency components of the murmurs, as well as of the second heart sound (the only one present), is quite obvious. Systole and diastole may be timed accurately on the stethogram by recording the electrocardiogram simultaneously. The nature of the mechanism producing the records will also be discussed later.

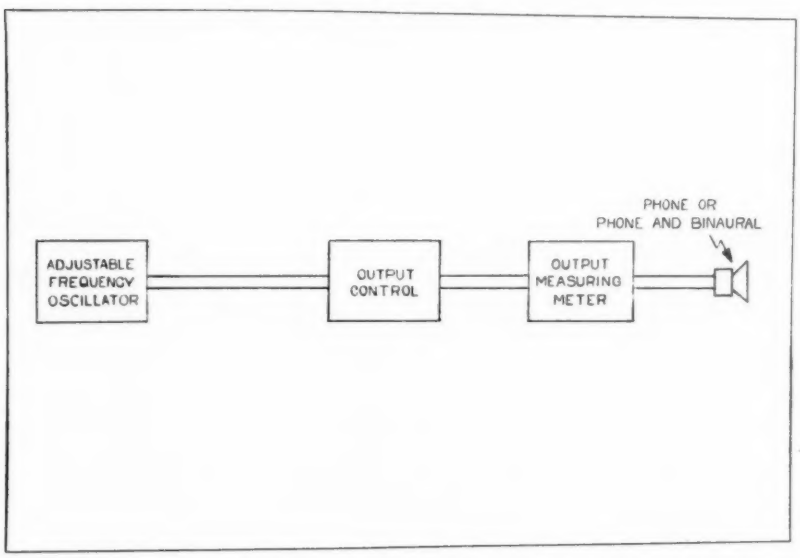

Fig. 7.—Schematic diagram of apparatus employed in determining the binaural effect.

Binaural Effects.—An experiment was performed in order to ascertain the effect of a binaural system in a stethoscope. Fig. 7 is a schematic diagram of the apparatus employed. An extremely quiet room was selected for the test (noise would definitely affect the accuracy of the test because it would alter the threshold of hearing).

The apparatus consisted of an adjustable frequency oscillator which was capable of producing a sinusoidal electrical wave practically free of any harmonic content. The frequency was adjustable over the auscultatory range. The electrical energy output at the various frequency settings was adjustable by means of a continuously variable control. The measured sinusoidal electrical waves were fed into a phone which could be placed to the ear of the person under test, or led to the ears through a binaural attachment.

The phone was applied to the patient's ear with a constant pressure throughout the test. For each frequency setting throughout the auscultatory range, the person who was being tested adjusted the output control to the point where the note was just audible, and the energy entering the phone was then measured. The same procedure was followed for the same phone with a binaural attachment.

If the resultant curves were plotted in decibels against frequency in cycles per second, we would have to take into consideration the characteristics of the phone, but, if the phone curve is subtracted from the binaural curve, the characteristics of the phone are canceled, and we obtain a direct ratio of hearing by the two systems. The reference level was taken as zero decibels, so that, if

any portion of the curve showed a positive decibel reading, it was an indication that the binaural system was more efficient by whatever the decibel reading happened to be; when the curve was negative, the monaural system was more efficient. Fig. 8 shows the resultant graph.

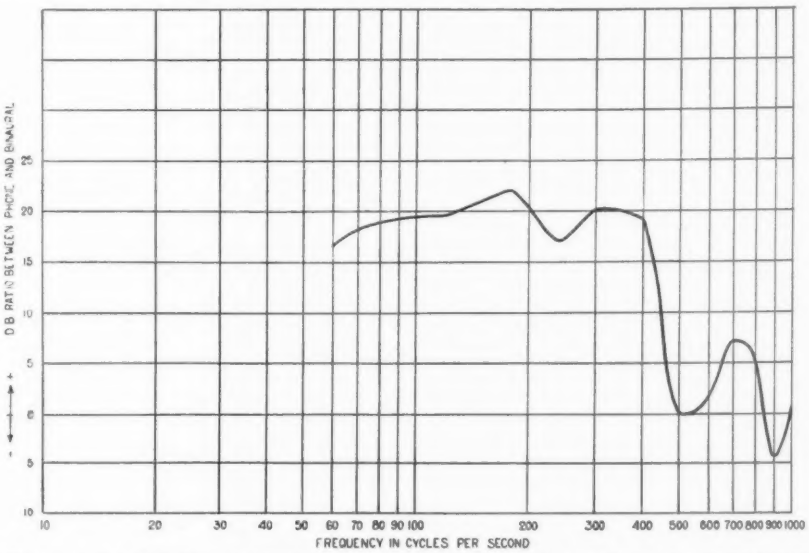

Fig. 8.—Relationship between monaural and binaural hearing in auscultation. Monaural hearing may be considered the equivalent of direct auscultation, whereas binaural hearing takes in the additive effect of both ears, minus the loss in efficiency of the stethoscopic attachment.

The following are some of the important relationships that exist between the monaural and binaural hearing systems in auscultation:

A. Direct telephone receiver application may be considered similar to direct auscultation.

B. A binaural system introduces resonance peaks, as may be seen in the graph.

C. In the range from 60 to 400 cycles per second, which includes most of the auditory range, the binaural system is, on an average, 20 decibels better than the monaural; this is equivalent to a tenfold increase in sound pressure at the eardrum at the threshold level.

D. Only between 850 and 1,000 cycles per second is monaural or direct auscultation more efficient than binaural, and this range is too high to be useful in auscultation.

Effects of Tubing Dimensions.—An important consideration pertaining to stethoscope performance is the length and caliber of the rubber tubing. It was previously mentioned that the pressure variations at the ear, produced by the motion of the skin or bakelite diaphragm, are inversely proportional to the internal volume of the stethoscope, so that an infinitely small volume would theoretically exhibit a maximum variation in pressure, which, in turn, would manifest itself as a sound of maximum intensity. Obviously, therefore, the tubing should be as short as possible, and the caliber as small as possible. The wall of the tubing should be sufficiently

rigid for maximum efficiency, for any wall motion reduces the effective pressure variation transmitted to the observer's ear.

A factor that must be taken into consideration is the frictional resistance offered to the air column by the walls of the rubber tubing. In other words, the efficiency of a stethoscope decreases as the resistance to the pressure variations is increased; the resistance is increased as the caliber is decreased. In order to eliminate the resistance component we have still to consider that the greater the volume, the less the efficiency. Therefore, to obtain the most efficient tubing for a stethoscope, one should make the tubing as short as possible and compromise on the resistance and volume components. The compromise may be approached by plotting a graph representing efficiency versus volume effect, and another representing efficiency versus resistance effect; where the two curves intersect is the point of optimum efficiency.

A test was performed to ascertain the actual change in efficiency of a binaural system caused by lengthening the rubber tubing. The apparatus employed in the experiment was identical with that shown in Fig. 7. The conditions under which the testing was done were the same as when the "Binaural Effect" experiment was performed. The only difference in the entire test was that a threshold of hearing curve (plotted in decibels input to the telephone receiver to produce the threshold of hearing level against frequency in cycles per second) was obtained for the phone and binaural attachment for various lengths of tubing. Two tubes were used to connect the binaural to the phone (audiphone, which will be described later). Fig. 9 shows the curves obtained with the three sets of tubing, which were 26 inches, 12 inches, and 3 inches long, respectively.

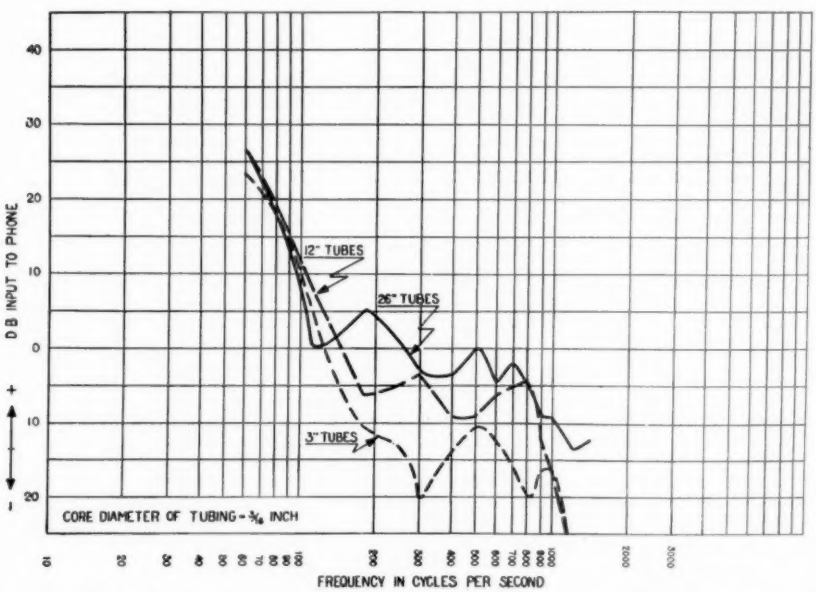

Fig. 9.—Curves illustrating transmissional losses in a binaural stethoscope as a result of increased tubing length.

The following are the general conclusions that may be drawn from a comparison of the three curves:

A. Below about 100 cycles per second, the efficiency is not affected materially by tubing length.

B. From about 100 cycles per second to 1,000 cycles per second, tubing length exhibits a considerable effect upon the efficiency; that is, the efficiency decreases with increased tubing length.

The changes in the efficiency of an acoustic stethoscope which are caused by varying the length of the tubing, although they are not given any consideration by stethoscope users, produce an effect upon the quality of the heart sounds. For example, at 200 cycles per second there is a 15-decibel attenuation of the sounds when two rubber tubes, each 26 inches long (commercial stethoscope tubing, with a caliber of $\frac{3}{16}$ inch) are substituted for two similar tubes 3 inches in length. The sound would be eight times as loud in the short tubing system. This attenuation occurs in the region where the low intensity, high-pitched diastolic murmurs are present, and every possible increase in efficiency in this region is of utmost value.

VI. Combined Amplifying Stethoscope, Stethograph, Electrocardiograph, and Sphygmograph

Introduction.—From the material presented thus far, in which the factors involved in auscultation are considered from a physiologic and physical point of view, we may safely conclude that the human ear, when operating in conjunction with an acoustic stethoscope, has many faults. In this paper several modifications in the design of the acoustic stethoscope have been suggested, with the purpose of increasing the auditory sensation efficiency of some of the sounds which are difficult to register. No doubt exists that, in the future, additional minor improvements may be developed in the acoustic stethoscope. However, after a thorough study of the physical behavior of the acoustic stethoscope, it appears that there is little room for radical improvements because of the inflexible properties common to the instrument as well as to human hearing.

Such a prediction concerning the future of the acoustic stethoscope appears rather bold, but, if average hearing is an unalterable constant, there remains only the acoustic stethoscope as the adjustable compensator for the defects of normal hearing. The physical make-up of an acoustic stethoscope is not very flexible; this is true of most pure acoustic systems. Until recently, a somewhat similar situation existed in the science of acoustics. Very little progress could be made in the study of acoustics, as well as the psychology and physiology of hearing, beyond a certain point, because of the inflexibility of the purely acoustic or acoustic-mechanical instruments which were available. This situation is well described in the recently published text "Hearing—Its Psychology and Physiology,"[23] by Dr. Stanley Stevens and Dr. Hallowell Davis, of Harvard University. With the adaptation of electronics to acoustics and the allied studies in the psychology and physiology of hearing, a new approach to the subject has been opened.

In a similar manner, the application of electronic, electromagnetic, and piezoelectric devices to auscultation has made possible a practically unlimited degree of freedom in the control of the over-all performance of pick-up device and ear, whereas, in the case of the purely acoustic stethoscope, flexibility in the control of this performance is limited. That is, there is sufficient freedom of adjustment in these more modern devices to allow certain modifications to be made in order to compensate for some of the auscultatory failings of the human ear.

In spite of the fact that such unlimited compensatory adjustments may be made, with certain decided improvements in the form of increased audibility of some physiologic chest and heart sounds, some of the inherent shortcomings of hearing cannot be overcome. This problem is summed up by Wiggers,[24b] as follows:

> "Most of the vibrations of the chest wall inaugurated by the two sounds of the heart have an amplitude and frequency which approach the lower auditory limit; indeed, it is probable that some of the vibrations are incapable of being recognized by the ear. While the auditory appreciation for slow vibrations of low frequency lasting only for brief moments can be cultivated by practice and training, the average ear and brain have some difficulty in differentiating the finer differences in time, frequency, and form, shown to exist in graphic records. When waves of several frequencies intermingle, the ear, according to inclination or training, tends to pick out one group and to suppress the other."

In addition to what we may consider the normal faults of the human ear as regards auscultation, another important condition must be taken into consideration. Many physicians who are under the impression that their hearing is normal will be found to have an audiogram which shows peaks and valleys in the auscultatory frequency region. The audiogram valleys in many cases fall well below the threshold of hearing for some of the important sounds. This characteristic of hearing has a tendency to become more exaggerated as the physician ages. The result of such changes is that certain sounds become inaudible to the doctor without his being aware of the fact.

Such shortcomings of hearing, whether normal or subnormal, may be surmounted by supplementing auscultation with graphic recording. A graphic recording system designed especially to record the required frequency band is immune to the auditory defects of the ear.

History.—At the turn of the century, attempts were made to improve auscultation by means of acoustic and electroacoustic devices. The experiments as a whole ended in failure when purely acoustic means were employed. An electroacoustic system was devised which did succeed in amplifying the sounds, but excessive distortion made the instrument useless. The electroacoustic stethoscope operated on a principle similar to that of the telephone; it consisted of a carbon-granule microphone which modulated an electric current, and this, in turn, excited a telephone receiver.

Successful amplification of the sounds was made possible by the development of the audion tube. Although the history of electronic amplification dates back to the practical results obtained by de Forest[25] in 1907, the audion tube amplifier was not sufficiently reliable for practical auscultatory application until a few years ago.

One of the earliest, practical, amplifying stethoscopes was developed by the Western Electric Company[26, 27] in 1924. Several other experimenters produced electronic-type amplifying stethoscopes. Since these devices altered the quality and character of the sounds so much that auscultatory technique had to be relearned, their clinical usefulness was decidedly limited.

Investigators such as Battaerd,[28] Brömser and Frank,[29] Crehore and Meara,[30] Einthoven,[31] Geluk and Einthoven,[32] Fahr,[33] Frank,[34-38] Gerhartz,[39] Hermann,[40] Lilienstein,[41] Myres,[42] Ohm,[43] Wiggers and Dean,[44] Williams,[45] Sell,[92, 93] Trendelenburg,[94-96] and others, realizing the clinical limitations of human hearing, made the earlier attempts at recording the heart sounds.

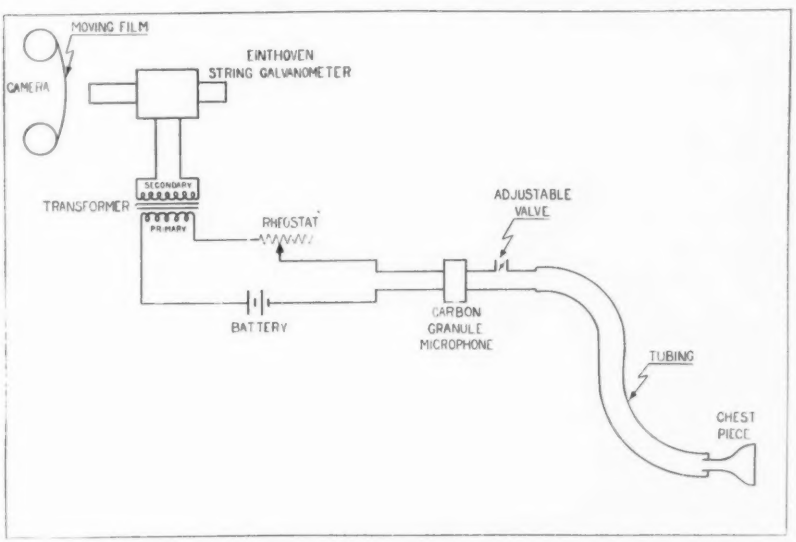

Fig. 10.—Schematic sketch of Einthoven's phonocardiograph.

One of the earliest types of recording stethoscopes that had some practical value was the Einthoven phonocardiograph. Fig. 10 shows a schematic sketch of the apparatus. A typical stethoscope chest piece was employed for accumulating the sounds, which were directed to a carbon-granule microphone. Interposed between the chest piece and the microphone was an adjustable leak valve, which was used to regulate the amount of low-frequency heart sound component reaching the microphone. The microphone performed the function of converting the minute sound pressure variations in the tubing into electrical pulsations whose intensity was adjustable by means of the rheostat; these electrical pulsations were allowed to pass through the primary winding of the transformer. The transformed electrical

pulsations were led off the secondary winding of the transformer and passed through the metallic-coated quartz string of the galvanometer. The purpose of the transformer was to prevent the direct current polarizing potential, produced by the battery which operated the microphone, from passing through the galvanometer string. The tension of the galvanometer string was greater than that employed in making electrocardiograms; this allowed the string to record the higher pitched sounds. The oscillations of the string were photographed in the usual electrocardiographic manner, thereby producing a phonocardiogram, as Einthoven preferred to call the graphic sound record. Einthoven could listen to the sounds by placing a telephone receiver across the secondary winding of the transformer.

Although the Einthoven phonocardiograph was capable of recording the heart sounds graphically, it was far from being a satisfactory apparatus for the following reasons:

A. Einthoven could not produce a smooth base line when no physiologic sounds were present, because extraneous noises entered the system through the leak valve. The undamped carbon particles of the microphone also produced extraneous noises.

B. The transformer employed by Einthoven introduced considerable distortion; understanding of audio-transformer performance was very limited at that time.

C. The sounds heard by placing a telephone receiver across the secondary winding of the transformer were so distorted as to be hardly recognizable.

Einthoven, although decidedly handicapped by the lack of suitable apparatus, did succeed in obtaining much valuable information concerning the nature of heart sounds.

The earlier investigators met with some success in registering heart sounds by the direct method, which is a system employing the physical principles of the human ear. The apparatus is usually composed of a stethoscope bell connected to a sensitive membrane by means of a rubber tube. The movements of the membrane produced by the cardiac sounds are recorded by means of an optical system focused upon a moving photographic film. The membrane in the direct system of heart sound recording may be compared with the tympanic membrane of the ear and is usually constructed of either thin rubber, mesentery from the guinea pig, gelatin, soap film, isinglass, or other such materials.

One of the most successful early stethographs was devised by H. B. Williams. It consisted of an electromagnetic telephone receiver operating in reverse as a microphone. The minute electrical pulsations were then intensified by means of a four-stage audion amplifier and recorded with an Einthoven string galvanometer. The Western Electric Stethophone[19] operated on essentially the same principle as the Williams apparatus. Trendelenburg's stethograph employed a condenser type of microphone, and Sell's apparatus employed an electrodynamic type of microphone, operating with a multistage amplifier and a moving-coil galvanometer. In recent years the piezoelectric crystal has been used as an element of the heart sound microphone.

The improvements incorporated by Williams, Sell, Trendelenburg, the Western Electric Company engineers, and others, although notable, did not succeed in reproducing the heart sounds with the fidelity with which they could be heard with the ordinary acoustic stethoscope or by immediate auscultation. The relationship between hearing and recording was extremely vague.

We shall now describe an apparatus that reproduces sounds without the slightest noticeable alteration in quality, and a method of recording the sounds simultaneously with the electrocardiogram and sphygmogram.

General Considerations.—An amplifying stethoscope, as the name indicates, is an apparatus which is capable of increasing the intensity of sounds; this may be accomplished either electrically, piezoelectrically, electronically, acoustically, mechanically, or electromagnetically, or by any combination of these methods. The following are some of the more important factors that must be taken into consideration in designing an amplifying stethoscope:

1. The apparatus must be capable of reproducing sounds without altering their character.

2. The amplifying stethoscope must be free of any inherent noise and be immune to the influence of external electrical wiring, diathermy machines, x-ray apparatus, etc.

3. Normal room noises and vibrations should not affect the operation of the apparatus.

4. The intensity of the sounds should be controllable by means of an easily adjustable, calibrated volume control.

5. The apparatus must be free of feed-back howls and squeals when the chest piece or sound pickup unit is brought into the vicinity of the hearing device.

6. The apparatus must be capable of standing rough handling.

7. It is advantageous to make the apparatus independent of house current, for the proper current and voltage are not universally obtainable.

8. The frequency response characteristic of the amplifying stethoscope should be of such a nature as to overcome, whenever possible, the natural human hearing defects which interfere with auscultation.

The stethograph, or heart sound recording mechanism, must be capable of performing the following functions:

1. It must record graphically all of the sounds that are audible with the stethoscope.

2. It must be capable of recording sounds that are easily missed as a result of masking and other phenomena caused by natural defects of the human ear.

3. When there are no sounds, i.e., during normal systole and diastole, the base line must be reasonably smooth.

4. The stethograph mechanism must be immune to all types of external electrical interference.

The electrocardiograph must be capable of satisfying all of the accepted standards and requirements. The electrocardiogram should be recorded simultaneously with the stethogram, in order to time properly the events of the cardiac cycle. Some stethograms are meaningless or confusing without a simultaneously recorded electrocardiogram or sphygmogram; this point will be illustrated later.

General Component Relationships.—Fig. 11 is a schematic illustration of the interrelationship between the various components of an apparatus which is capable of producing, simultaneously, an electrocardiogram-stethogram and amplified heart sounds.

The amplifying stethoscope component consists of a microphone which serves to convert the sound energy picked up at the chest wall into equivalent, minute, electrical pulsations. These minute electrical pulsations are passed into an electron-tube amplifier, and the amount of amplification is regulated by a volume control device. The audiophones, which are connected to the output of the amplifier, reconvert the strengthened electrical pulsations into sound energy which is identical with that picked up by the microphone.

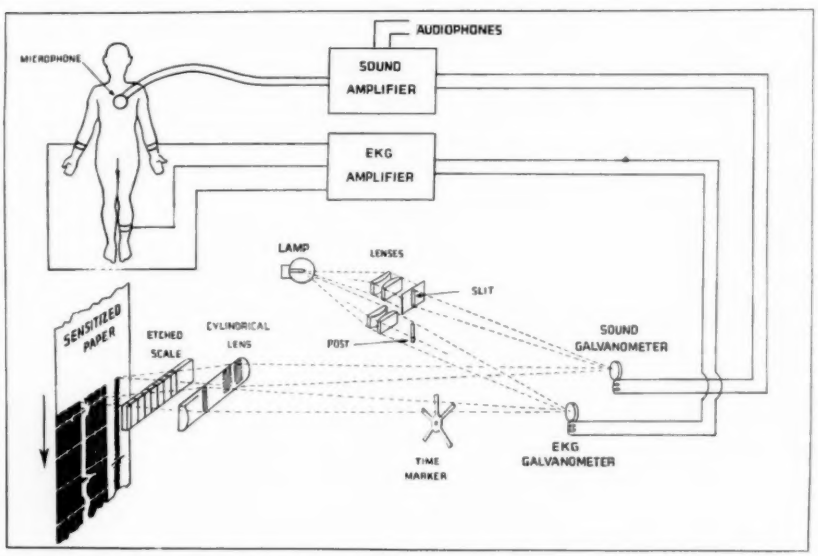

Fig. 11.—Schematic diagram illustrating the interrelationship of the various components of an apparatus capable of producing a simultaneous electrocardiogram-stethogram and amplified heart sounds.

A moving-coil (d'Arsonval) galvanometer which is capable of responding to the frequency band of the sounds encountered in auscultatory work is also connected to the output of the sound amplifier. The galvanometer coil, which carries a mirror, oscillates in response to the intensified electrical pulsations. A beam of light is reflected from the mirror upon moving, sensitized film or bromide paper. The oscillation of the mirror moves the light beam across the paper. The combination of the longitudinal motion of the paper and the transverse movement of the light beam produces a graph of the sounds, plotted against time (a stethogram).

The electrocardiogram is obtained by directing the cardiac currents into a proper switching and standardizing system, amplifying or strengthening the minute currents, and feeding them to another moving-coil (d'Arsonval) galvanometer whose frequency characteristics are suited to electrocardiography. By means of a suitable optical system, the movements of the galvanometer coil are recorded on the same sensitized paper or film upon which the heart sound beam falls, thereby producing a simultaneous electrocardiogram-stethogram. Vertical time marking lines, together with horizontal amplitude lines, are superimposed optically over the electrocardiogram section, which occupies 4 cm. of a 6-centimeter-wide film or paper; the remaining 2-centimeter band is devoted to the stethogram.

The Microphone.—The invention of the telephone and the successful transmission of a complete sentence by Alexander Graham Bell in 1876 marked the beginning of the development of devices capable of converting sound energy into electrical energy. The development of the fundamental principles of nearly all microphones (technically known as electroacoustic transducers) which are in use today dates back to Bell's time, but, until recently, the microphones could not be used because they were not sensitive enough. The perfection of the vacuum-tube amplifier in recent years has made it possible to take advantage of many of the desirable properties of these electroacoustic transducers.

A microphone is an electroacoustic transducer which, when actuated by the power in an acoustic system, will deliver power to an electrical system, and the wave form in the electrical system will correspond to that in the acoustic system.

When Einthoven performed his experiments with the phonocardiograph, he did not have an electronic type of amplifier. As a result, he was forced to employ a microphone which was not dependent upon electronic amplification. The best transducer available was the carbon-granule microphone.[48, 49] As previously observed, this type of transducer, when employed for stethographic purposes, possessed many limitations. H. B. Williams improved upon the Einthoven system by substituting a magnetic type of telephone receiver to whose diaphragm a nonmetallic button was cemented. The button was placed in contact with the patient's chest, so that the sound vibrations were transmitted mechanically to the diaphragm. The vibrating diaphragm, in turn, induced an equivalent electromotive force in the coil of the electromagnet of the telephone receiver. The coil was electrically connected to a step-up transformer which fed into an amplifier.

Although Williams' microphone was a decided improvement over the one employed by Einthoven, in that it eliminated the background noise caused by the carbon granules, it possessed some undesirable qualities, as follows:

1. The Williams microphone required a coupling transformer between the microphone and amplifier. If a transformer is followed by a highly sensitive amplifier, minute alternating current interferences may be picked up from electrical appliances by the transformer, amplified, and superimposed upon the heart sounds.

2. When the ear piece (the device which reconverts the amplified electrical pulsations into amplified sounds) was not kept well away from the microphone, an electromagnetic feed-back took place and a howling sound was heard.

3. Considerable difficulty was encountered in subduing extraneous noises caused by slight shifting of the microphone contact button on the patient's chest, unless the microphone was carefully fastened down with adhesive tape. This is a rather awkward clinical procedure.

Sell, in an endeavor to improve upon the Williams apparatus, constructed an electromagnetic microphone. It consisted of a magnet and a movable coil. The coil was physically connected to a button which was in contact with the patient's chest. A double-walled, suction type of funnel surrounded the microphone button. The microphone was operated by suspending the unit from a cushioned pulley, and fastening the suction bell to the patient's chest. This arrangement placed the microphone button in contact with the chest and thereby provided for transmission of the sound vibrations to the coil. The vibrating coil, in turn, cut the lines of force produced by the fixed magnet, setting up an electromotive force in the coil. The coil was electrically connected to a transformer, followed by an amplifier, as in the case of the Williams apparatus.

The major improvement in the Sell microphone was in the construction of the chest piece, which eliminated to a large extent the extraneous noises caused by slight shifting of the button on the chest. The other undesirable features of the Williams microphone were also present in the Sell microphone, for the general nature of the two was the same.

The suction bell functioned very well when there was no heavy growth of hair on the chest. However, in cases in which hair was present, the suction could not be maintained, and the chest had to be shaved. The application of an oily substance, such as vaseline, on the patient's chest often helped maintain the vacuum if the hair was not dense.

Trendelenburg devised a microphone which operated on the electrostatic principle. It is better known as a condenser type of microphone. In its simplest form, a condenser microphone comprises a light, stretched, metallic diaphragm, spaced at a very short distance from another, parallel, metallic plate, which acts as a fixed electrode. A steady, direct-current, polarizing potential, such as that used with the carbon-granule microphone, but of considerably higher value (usually about 135 volts), is placed across the parallel plates through a series resistance. The minute, alternating variations of capacity caused by the movements of the diaphragm under heart sound excitation create a variable impedance which produces an alternating current that is equivalent in character to the heart sounds. The generated alternating current flowing through the resistor, which is connected in series with the microphone, sets up an equivalent alternating potential across the resistor, and this is fed to an amplifier and intensified.

The Trendelenburg microphone employed a suction bell similar to that of the Sell microphone, with the exception that the chest wall motions were transmitted to the diaphragm through an air column instead of by direct mechanical contact. Trendelenburg's microphone was far superior to the previously mentioned types, in that it eliminated the electromagnetic feed-back howl when an electromagnetic ear piece was employed. However, two major faults still remained, namely:

1. The characteristically low sensitivity of a condenser type of microphone necessitates the use of an amplifier possessing considerably more amplification and a higher degree of instability.

2. When relatively high pressures are suddenly set up in the air column of a condenser microphone, for example, when the bell is applied to the patient's chest, the diaphragm tends to be forced over to the other plate electrode. Because there is a polarizing potential between the two electrodes of the microphone, pitting of the metal takes place at the point of contact every time the electrodes touch. After a number of such contacts, the microphone becomes inoperative.

The electrical impedance of a microphone may vary anywhere between a few ohms and many hundreds of thousands of ohms, depending upon the type. For satisfactory operation, the electrical impedance of a microphone must match the impedance of the following circuit element. In the case of an electromagnetic type of microphone, such as that of Sell or Williams, the impedance of the coil must be properly matched. The impedance of such a coil (depending upon the design) may vary between a few ohms and several thousand. Therefore, in order to match such an element to an amplifier and retain maximum efficiency and a minimum of distortion, a coupling transformer must be introduced in such a way that the primary winding of the transformer matches the microphone coil, and the secondary winding of the transformer matches the amplifier.

In the case of a condenser microphone, such as the Trendelenburg type, the inherent impedance is extremely high, and of the same order as the input of an amplifier. As a result, no coupling transformer is necessary. Thus, some of the previously mentioned difficulties which are encountered when a transformer is placed in so delicate a location may be eliminated.

During the last few years the radio manufacturers have perfected the piezoelectric crystal microphone. A crystal microphone is one in which the electrical output is dependent upon the stresses set up in a crystal

of rochelle salt, quartz, or tourmaline. In such a device the output potentials are proportional to the stresses which the sound waves induce. Rochelle salt crystals are most commonly used in crystal microphones because the piezoelectric constant of rochelle salt is more than one thousand times that of quartz and tourmaline.

The piezoelectric crystal element possesses many characteristics which make it suitable for stethography; the following are some of the most important:

1. The crystal element is inherently free of noises.

2. The crystal element is small and light and requires only electrostatic shielding.

3. It does not require any polarizing potentials.

4. The impedance of a crystal element is of such an order that a coupling transformer is not required.

5. The output level (voltage output vs. excitation) is quite high as compared with other types of microphone elements.

6. The crystal element is comparatively rugged.

When all of the advantageous properties of the crystal microphone are considered, it will be understood why such an element was chosen for our microphone.

A detailed technical discussion of the design and mathematical theory of crystal microphones which are suitable for stethographic, as well as auscultatory, work is obviously beyond the scope of this paper. However, a general consideration of some of the more important features should be of interest.

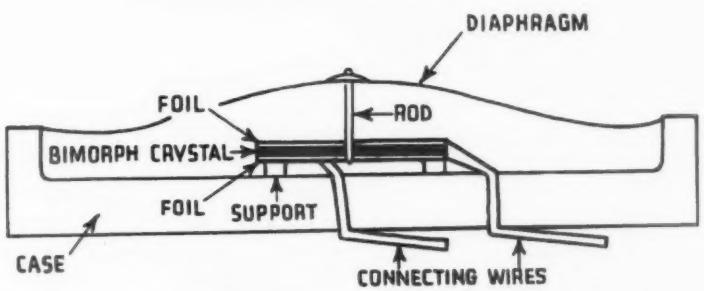

Fig. 12.—Cross-section sketch of the crystal microphone cartridge.

The heart of the microphone is a cartridge or diaphragm type of crystal unit (Fig. 12). It consists of a curvilinear diaphragm which is directly coupled to a bimorph rochelle salt crystal through a tiny connecting rod; the crystal and connecting rod are located in the interior of the cartridge. Should an actuating force be applied to the diaphragm, proportional stresses will be set up in the crystal, and it will transform these stresses into equivalent electrical potentials which are conducted away from the crystal by means of metallic foil electrodes in contact with it.

The fundamental frequency, or natural period, of the bimorph crystal is approximately 10,000 cycles per second. When a diaphragm is coupled to the crystal

(Fig. 12), the fundamental frequency of the combination is lowered to a few thousand cycles per second. This is still well above the upper frequency limits encountered in auscultatory work.

Fig. 13*A*.—Microphone case, with built-in, acoustic, high-pass filter, and piezoelectric cartridge.

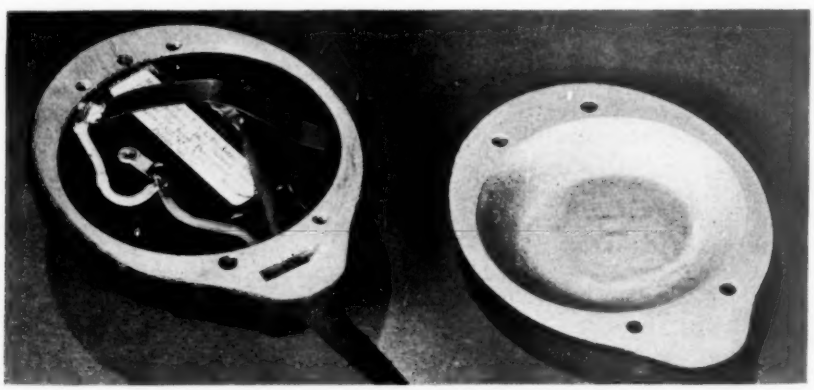

Fig. 13*B*.—Manner in which cartridge is inserted into microphone case.

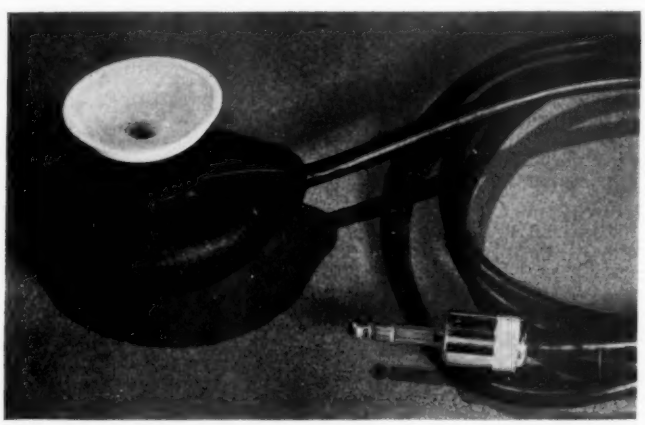

Fig. 13*C*.—Completely assembled microphone, with a large, open chest piece.

The cartridge is fitted into a case especially designed for stethography (Fig. 13*A*, *B*, *C*). The microphone case proper is constructed to incorporate what is technically known as an acoustic high-pass filter. The filter serves two essential purposes,

namely: (1) It eliminates the extremely low-frequency, chest wall motions which have no known physiologic significance. (2) It protects the crystal from sudden, high pressures of extremely low frequencies, such as those which are produced by applying the microphone to the chest.

A filter, whether it be electrical, mechanical, or acoustic, is a system which will freely pass desired frequency bands, and highly attenuate neighboring, undesired bands. Campbell[57] is credited with having developed the electrical type of filter; Stewart[58] devised the first acoustic types. The mechanical type of filter has been employed in various kinds of apparatus for many centuries. The electrical wave-filter action has been further investigated by Zobel,[59] Johnson,[60] Shea,[61] and others; the acoustic filter, by Mason[62]; and the mechanical types, by Crandall.[63] The most complete investigations on filtering action have been carried out for electrical types. However, the analyses of the acoustic and mechanical types of filters are applicable and are very similar to those of the electrical types.

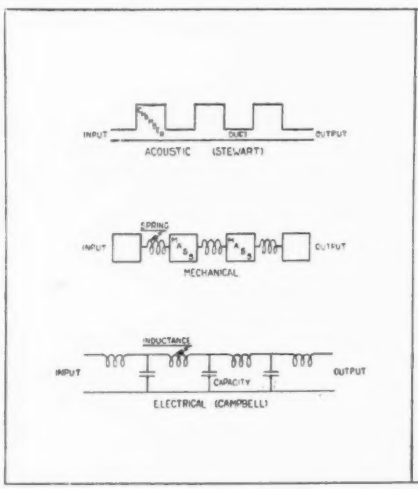

Fig. 14.—Equivalent low-pass filters.

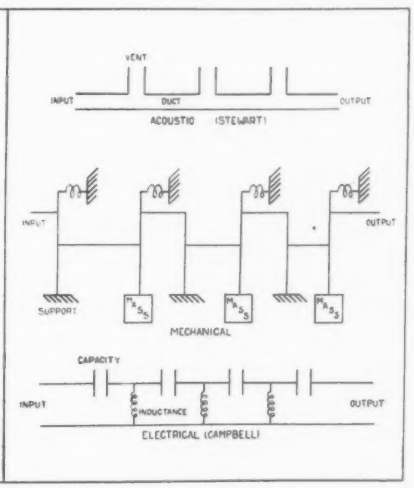

Fig. 15.—Equivalent high-pass filters.

Fig. 14 shows three equivalent filters which possess low-pass characteristics. These filters are known as two-element types because each element contains two components. The condenser in the electrical circuit, the chamber in the acoustic filter, and the spring in the mechanical filter perform corresponding functions. The inductance in the electrical filter performs a function similar to that of the duct in the acoustic filter and the mass in the mechanical filter. Fig. 15 shows three equivalent filters which have high-pass characteristics. Theoretically, a low-pass filter should allow the passage of electrical currents, velocities, and volume currents, in electrical, mechanical, and acoustic systems, of all frequencies lying between zero and the filter cutoff frequency, which is dependent upon the constants of the filter. On the other hand, a high-pass filter will allow the passage of currents, velocities, and volume currents, in electrical, mechanical, and acoustic systems, of all frequencies between the cutoff frequency and everything above it to infinity.

The carbon-granule microphone employed by Einthoven in his phonocardiograph possessed a simple, but crude, sort of high-pass acoustic filter. Einthoven accomplished the filtering process by introducing an adjustable valve (Fig. 10). The fact that Einthoven allowed access between the external air and the air column which was under heart sound excitation was one of the main causes for the introduction of extraneous noises.

In our crystal microphone, the acoustic filter channels are so constructed that they connect with a chamber hermetically sealed into the microphone case; the chamber acts as a substitute for the external air. This type of acoustic filter design has made possible the necessary degree of high-pass filtering action upon the heart sounds, without introducing the slightest traceable extraneous noise.

The four bells, or chest pieces, of Fig. 3 are used in conjunction with the microphone. Each bell, as previously pointed out, produces a different degree of filtering action. The desired bell may be screwed into the microphone case.

The Audiophone.—In an amplifying stethoscope, the audiophone is the component which converts the amplified electrical pulsations into equivalent sounds such as those which are normally heard with the acoustic stethoscope. Fig. 16 is a photograph of an audiophone. The three fundamental components of an audiophone are the electroacoustic transducer, the case, and the binaural.

The electroacoustic transducer portion of the audiophone is an electromagnetic telephone receiver of the diaphragm armature type. A telephone receiver is an electroacoustic transducer which, when actuated by an electrical system (the amplifier, in the case of a stethograph), will supply power to an acoustic system, and the wave form of the sound in the acoustic system will correspond to the wave form in the electrical system. Telephone receivers may be divided into four general classes, namely, (1) electromagnetic, (2) electrodynamic, (3) electrostatic, and (4) piezoelectric. Complete descriptions of the electromechanics of these electroacoustic transducers may be found in most good texts or handbooks on applied acoustics,[64] telephony,[48] and electrical engineering.[66] The electromagnetic type, which was invented by Alexander Graham Bell and perfected by others, was selected as the audiophone component because of its simple construction, rugged nature, ability to cover the required frequency band, compactness, and suitable impedance characteristics.

The telephone receiver first developed by Bell consisted of an electromagnet which retained a residual amount of magnetism. The strength of the residual magnetic field could be modulated by the electric signal which was allowed to pass through a coil wrapped around the magnet. A steel diaphragm, clamped at its circumference and placed a short distance from the pole pieces of the electromagnet, was caused to vibrate at a frequency and to a degree which depended upon the frequency and strength of the signal current. The diaphragm action, in turn, set the surrounding air into motion, and this produced the sound which was perceived by the ear. The present-day instruments operate on the same principle, but improved materials and design have greatly increased their efficiency and sensitivity.

The only moving part in an electromagnetic receiver of the diaphragm armature type is the diaphragm. Although the system appears extremely simple from a mechanical standpoint, the theoretical analysis is very complex. Theoretical investigations of the apparatus have been made by Crandall,[63] Kennelly,[66] Lamb,[67] and several others. All seem to agree that the mechanics of the diaphragm resembles very closely that of an ideal, clamped, circular plate.

In the design of the electroacoustic transducer which is employed in the audiophone, a diaphragm with a fundamental resonance peak of roughly 1,000 cycles per second was selected. Following this resonance point, other resonances occur as the frequency is increased. The fundamental resonance and certainly the higher frequency modes of vibration are well above the auscultatory frequency band and, therefore, produce no ill effects.

The acoustic system in an audiophone consists of the inertance produced by the binaural system and the capacitance of the volume which is excited by the diaphragm. In an analysis of the action of an audiophone, the acoustic system, as well as the mechanics of the diaphragm, must be considered.

In an ideal audiophone, the ratio of sound pressure variation delivered to the ear cavity to the voltage applied to the transducer should be independent of the frequency. If we make the assumption that the cavity of the ear presents a constant

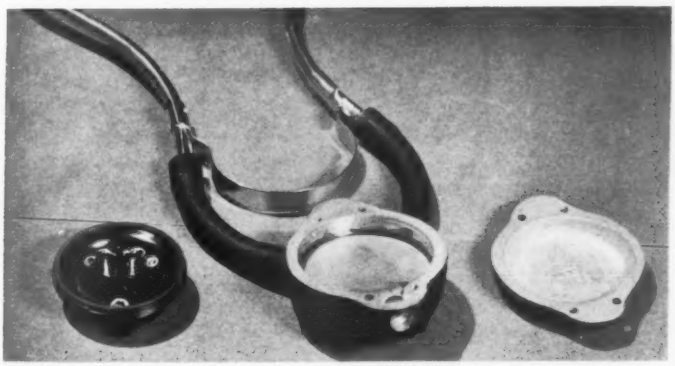

Fig. 16*A*.—Audiophone, with cover and electromagnetic phone removed.

Fig. 16*B*.—Manner in which electromagnetic phone is inserted into audiophone.

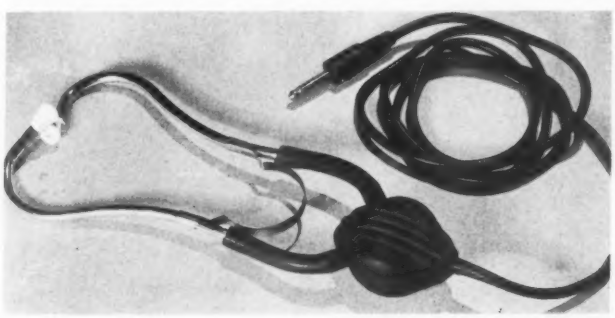

Fig. 16*C*.—Assembled audiophone.

capacitance to the audiophone, the ratio of the pressure developed in the ear cavity to the amplitude of the diaphragm vibrations should be independent of the frequency.

An interesting point is that the impedance offered by the ear cavity to the audiophone at the higher frequencies is not purely capacitive, but becomes more so at the lower frequencies. This phenomenon is caused by the presence of standing wave systems between the transducer and portions of the ear cavity, as well as by absorption effects which may be considered equivalent to a resistive component. This effect is of small consequence in auscultatory work, for the frequencies which are encountered do not exceed 1,000 cycles per second. On the other hand, at low frequencies, leakage which may occur in an improperly designed audiophone will give rise to a reactive and resistive component. This factor may vary from person to person if the ear pieces of the binaural are not properly placed. It is an important consideration in the action of the acoustic, as well as the amplifying, type of stethoscope. An hermetically sealed audiophone also eliminates room noises which seem greatly exaggerated; the same holds true for the acoustic stethoscope.

The Amplifier.—The stethograph-electrocardiograph amplifier (Fig. 17) comprises two amplifier channels of entirely independent and different electrical characteristics. One channel possesses electrical characteristics which are useful for amplifying the heart and chest sounds, whereas the other channel is suitable for amplifying the electrical heart action potentials which are encountered in electrocardiography.

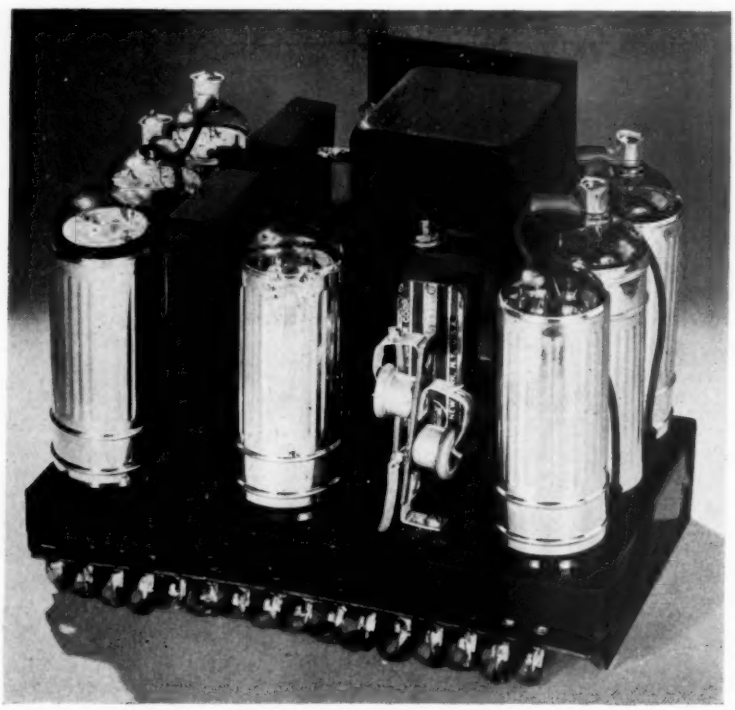

Fig. 17.—The stethograph-cardiograph amplifier.

The amplifier systems of the electrocardiograph-stethograph employ thermionic vacuum tubes. A thermionic vacuum tube consists of an evacuated enclosure containing a number of electrodes between two or more of which conduction of electricity through the vacuum may take place. The three most essential electrodes in a thermionic vacuum tube which is suitable for amplification purposes are the cathode, grid, and plate.

The grid is interposed between the cathode and the plate, and serves as a means of controlling the flow of current in the plate circuit. That is, electrons from the heated cathode normally flow between the cathode and the plate, and, if a potential is applied to the grid, making it negative with respect to the cathode, the current flowing between the plate and cathode is reduced by a proportionate amount. A given change in grid potential has a much greater effect in changing the plate current. If a resistor is placed in the plate circuit so that the plate current variations are allowed to pass through the resistor, a proportional voltage variation will take place across the resistor. Thus the voltage variation across the plate resistor is similar to the grid voltage variation but is amplified.

When a greater degree of amplification is desired than can be obtained with a single stage or tube, audion tubes may be combined to form a multistage or cascade amplifier. The tubes of succeeding stages of a cascade amplifier must be interlinked in proper sequence through some medium which will transfer the intensified signal from one stage to the other for additional intensification without affecting the operation of each individual stage.

The plate circuit in each tube of a multistage amplifier must be closed by some form of impedance or resistance; this is also true for a single stage. The signal potential that manifests itself across the impedance load is then impressed upon the grid of the next tube or stage. There are several classifications of multistage amplifiers according to the plate-load impedance circuit,[68] such as resistance-capacity, impedance-capacity, direct-coupled, and transformer-coupled.

The resistance-capacity coupled circuit employed in the stethograph amplifier comprises the arrangement in which the plate load is a pure, high resistance. The grid circuit of the succeeding stage is also composed of a pure, high resistance. The grid and plate resistors are linked by a condenser which acts as an isolating medium for the direct current potentials in the grid and plate circuits. Should the direct current potentials which control the operation of the amplifier stages intermingle, the amplifier as a whole would not function properly, if at all. The signal potential, which is of an alternating character, is capable of passing through the condenser.

The degree of amplification of the signal depends upon the amplification provided by the tubes in the various stages, in addition to whatever attenuation may take place as a result of the relationship of signal frequency to the resistance-capacity values employed for coupling. The relationship is commonly known as the time-constant, or frequency response characteristic, of the amplifier. The frequency response characteristic of the amplifier, as well as that of the audiophone and microphone, affects the quality of the heart sounds as heard with the combination or amplifying stethoscope.

The electrocardiograph amplifier channel operates on the same principle as the stethograph, in that both are resistance-capacity coupled. However, the electrocardiograph amplifier possesses a time constant which permits all frequencies encountered in electrocardiography to be amplified without the slightest degree of attenuation or accentuation.

Frequency Response vs. Quality.—A theoretical analysis of the relationship that exists between the frequency response characteristic of an acoustic or amplifying stethoscope and the quality of the sounds as heard by the observer is of utmost importance. A thorough study of all available literature on stethoscopes, whether of the amplifying, acoustic, or recording types, shows a surprising lack of reference to frequency response. A still more surprising fact is that there are no set

standards to follow; this is certainly a unique situation when we consider the importance of correct design in an instrument which is so widely used.

The primary requirements for any kind of stethoscope which is to be used by a competent observer with normal hearing are as follows:

1. The stethoscope must be capable of transferring all of the sounds from the patient to the observer's ear.

2. The transferred sounds must possess a quality or pitch of such a character as to be familiar to the observer.

3. The stethoscope must possess a means of attenuating certain portions of the auscultatory frequency band, in order to reduce masking effects.

In order to obtain a starting point in the effort to ascertain the optimal frequency response of a stethoscope, a study was made of the acoustic stethoscopes which are on the market. They were found to vary in tonal quality, and, in addition, the various commercially available chest pieces introduced different tonal qualities, as well as variations in intensity.

The next step was to investigate the nature of the two most commonly used chest pieces, namely, (1) the open bell, with a diameter of 1 inch, and (2) the "Bowles," or diaphragm type, with a working diameter of 1⅜ inches.

There was a slight variation in tonal quality and intensity when the most commonly used stethoscopes which employ these two chest pieces were compared. The general consensus of cardiologists was that, although a noticeable difference in tonal quality existed, it was insufficient to affect the general auscultatory technique.

Frequency response characteristic curves were obtained for the various popular makes of stethoscopes which employ the two chest pieces, and a mean frequency response characteristic was calculated. With this average curve as a basis, we constructed an amplifying stethoscope with a frequency response characteristic which was identical with the theoretically average stethoscope.

Fig. 18 is a schematic diagram of the apparatus employed in ascertaining the frequency response from which the mean stethoscope response characteristic was calculated. The characteristics of the loud speaker, the transmissional effects of the media comprising the patient's chest, and other such modifying factors were eliminated by the subtraction method (previously described in connection with the determination of the characteristic curves of the chest pieces of Fig. 5). A theoretical amplifying stethoscope frequency response curve, not including the modifying effects of the chest pieces which are capable of reproducing the sounds as they are heard with the theoretically average, acoustic stethoscope, is shown in Fig. 19. The construction of an amplifying stethoscope which has a frequency response characteristic identical with the theoretical one of Fig. 19 is quite practical, and this feature has been incorporated in the apparatus. It is well to mention that a slight variation in the slope of the curve below 200 cycles per second is sufficient

to alter considerably the quality of the sounds as they are heard with the amplifying stethoscope. A variation of considerable degree in the slope of the curve above about 200 cycles per second produces little effect upon the quality of the sound.

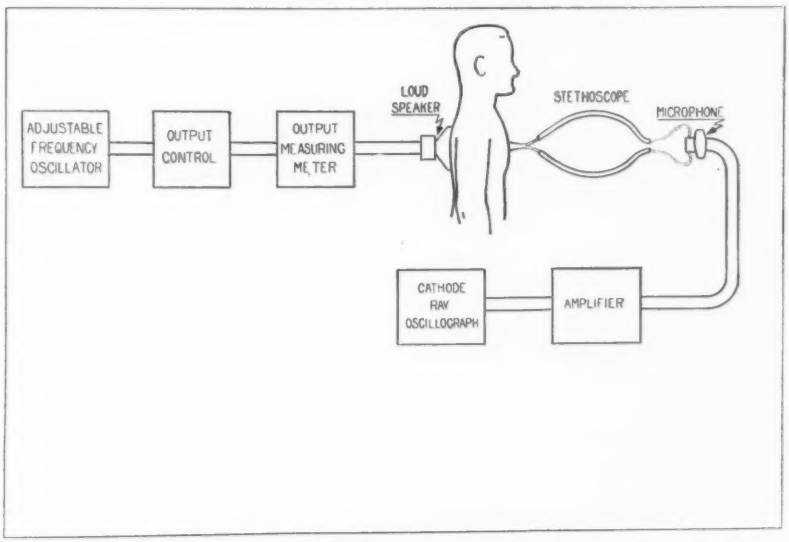

Fig. 18.—Schematic diagram of apparatus for comparing frequency response characteristics of acoustic stethoscopes and amplifying stethoscopes.

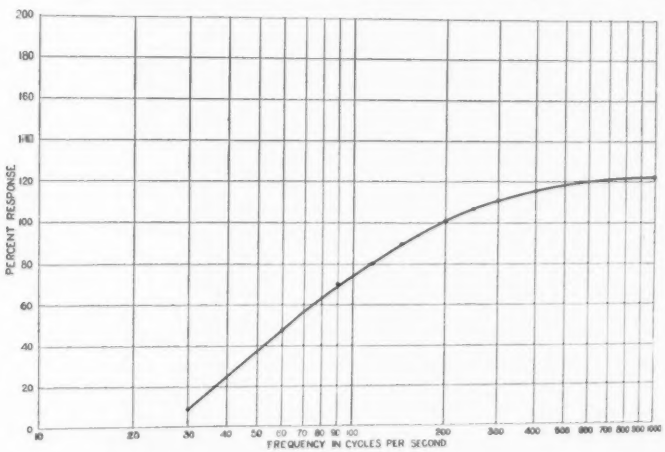

Fig. 19.—Output of amplifying stethoscope vs. input to microphone, omitting characteristics of chest piece. Ordinate plotted in per cent response instead of decibels, to accentuate graphically the degree of slope.

An amplifying stethoscope with a frequency response curve which is flatter below 200 cycles per second than the graph of Fig. 19 has a tendency to convey the sounds to the observer's ear (especially the low-pitched mitral diastolic murmurs) with a raspy effect. This foreign quality is so different from what is normally heard with the acoustic stethoscope that, at times, the observer has the feeling that the time he spent in perfecting his auscultatory technique was wasted, unless he

keeps going back to the acoustic stethoscope. This has been the major difficulty with electrical stethoscopes; it is no fault of the system, but of the basic design of the apparatus. *The low-pitched, raspy effect is more common in the electrical type of stethoscope than in the acoustic variety because it is easy to accentuate low-pitched sounds with an amplifying system, whereas, in the acoustic stethoscope, the low pitches are limited in relative intensity by the physical nature of the apparatus.*

Effects of Sound Intensity Variation.—Earlier in this paper it was shown that the average ear is most sensitive to variations in intensity above 50 decibels and gradually becomes less sensitive at the lower sensation levels. This is important in auscultation, for the intensity level is usually 10 decibels, or less, and the frequency band which is involved is at the lower extremity of the human hearing range. It is well to repeat that, with a frequency of 60 cycles per second, an intensity variation of 20 per cent is just perceptible when the sound level is 50 decibels, and a variation of as much as 200 to 300 per cent is necessary when the intensity level is 10 decibels.

Although a higher sensation level requires less of a frequency change for minimum perception, the ability of the ear to detect changes in frequency at the lower levels of intensity is not as poor as its ability to perceive intensity variations.

It has also been pointed out that the intensity of the sounds bears an important relationship to masking, pitch, etc. Stevens and Davis,[23] in their discussion of this phase of hearing, state:

"It has been the traditional view of psychology that the attributes of sensation show a one-to-one correspondence to the dimensions of the stimulus. Some such view is also implicit in the naïve epistemology of the physicist. He tends to think of pitch as if it were the perception of the frequency of a tone, but we have seen that holders of that view run into difficulties. The pitch of a pure tone can be altered without changing its frequency; likewise, the loudness of a tone may be varied without changing its intensity. Pitch is a function of two physical variables, frequency and intensity—loudness is a different function of the same two variables. Both pitch and loudness are fundamentally to be conceived as *reactions* on the part of organisms to sound waves. These are systematic reactions, to be sure, and can be ordered on scales and evaluated, but they are, nevertheless, products of the interaction of an atmospheric disturbance with a living system."

The authors further state:

"Several writers, during the last century, have disclosed the fact that tones are characterized by an apparent 'largeness' or 'extensiveness' (Rich[69]). The low tones of an organ appear to be 'bigger' than the high chirp of a cricket, even when the loudnesses of the two are equal. This subjective aspect of a tone is known as volume. (The radio-engineer speaks of the 'volume' of a sound, or of the 'volume control' of a radio set, but he means by volume what we should properly call intensity.)"

In order to illustrate the manner in which these characteristics of hearing are related to auscultation, let us assume that a certain heart

lesion is present in an emaciated person, an obese person, a heavy, "barrel-chested" man, and a person with a chest of normal proportions. Let us further assume that the intensity, pitch, and quality of the heart sounds and murmurs are identical at the source in each case. Because of the different transmissional characteristics in these cases, the intensity of the sounds, as they are heard on the surface of the chest with the same acoustic stethoscope, varies decidedly. As a result of the intensity modification alone, sounds that are audible in the case of the normal and emaciated persons may not be heard at all when the stethoscope is applied to the obese and "barrel-chested" patients.

If the acoustic stethoscope employed in these cases had an adjustable intensity control, and the intensity levels were the same in all (i.e., approximately that of the sounds heard in the case of the patient with a normal chest), a considerable percentage of the distortion, or modification effects, would be eliminated. Also, certain possible masking effects could be reduced by slightly varying the intensity above or below that of the sounds heard over the normal chest; a noticeable variation in quality and pitch would also be apparent at the various hearing levels. Obviously, the acoustic stethoscope does not lend itself to adjustable intensity control, whereas the amplifying stethoscope is capable of such control.

From this discussion it should be obvious that an amplifying stethoscope is not primarily an instrument to be used for making sounds many times louder than they can be heard with an acoustic stethoscope. The major advantage of the amplifying stethoscope over the acoustic stethoscope is that the intensity can be adjusted as desired, and thus a number of modifying characteristics which cannot be overcome with the acoustic stethoscope are eliminated. Roughly, the heart sound intensity in a normal young person, as perceived by auscultation with an acoustic stethoscope, is approximately optimal from a standpoint of masking, pitch, quality, and accustomed usage of the older acoustic stethoscope.

Because of deviations of the individual physician's hearing from the "Average Normal Threshold Curves of Audibility and Feeling" (Fig. 1), it is obvious that, in any given case, the sounds heard by a group of trained observers, employing the same acoustic stethoscope, will not register identically, although the hearing of each may be considered normal. The most marked variations from normal would be found in the older observers, whose hearing is not so sensitive as that of the younger physician. A slight increase in the sensitivity of the stethoscope, to compensate for the decreased sensitivity of the ear, will bring back the keenness of hearing which is so necessary for maximum efficiency. Such compensation is at present impossible except with an amplifying stethoscope which has an adjustable and calibrated intensity control.

Sound Filters.—An acoustic or electrical filter system may be used with the electronic or electrical amplifying stethoscope as an aid in minimizing masking effects. Acoustic filtering is accomplished by means of the open and diaphragm chest pieces, which can be attached to the piezoelectric, crystal microphone. Electrical filtering is obtained by inserting "Campbell" filters, or any of the well-known modifications of the "Campbell" filter, between the microphone and the audiophone.

Dodge and Frederick,[70] of the Western Electric Company, first employed the "Campbell" filter in an amplifying stethoscope. The filter system was composed of a series of high-pass and low-pass filters. By means of a suitable switching system, any filter or combination of filters could be inserted into the amplifier circuit to allow any desired low frequency band, high frequency band, or intermediate frequency band to pass to the translating device. The amplifier proper had a frequency response characteristic that was flat over the auscultatory range.

From the physicist's point of view, the Dodge and Frederick system may be considered an excellent instrument for analyzing the frequency components of sounds. Williams and Dodge[18] and Cabot and Dodge[22] utilized the apparatus as an auscultatory wave analyzer, with noteworthy results. On the other hand, the clinical use of the apparatus was limited by the fact that it altered the quality of the sounds which are heard with the ordinary acoustic stethoscope. The modification of tonal quality introduced by the Dodge and Frederick apparatus was caused by the fact that its frequency response characteristic was different from that of the acoustic stethoscope. Furthermore, the sharp cutoff characteristics of the "Campbell" filters produced additional modifications of the sounds, although the sharper the cutoff the more suitable is the apparatus as a sound-wave analyzer.

In our opinion, for optimum clinical results, a stethoscope, whether it be electrical or acoustic, should possess a frequency response characteristic similar to that of the average commercial acoustic stethoscope, minus the modifying effect of the chest piece. The filters, whether they be electrical or acoustic, should possess modifying frequency response characteristics similar to those obtained with open bells of various diameters and diaphragm chest pieces made of plastic materials. In other words, the function of a filter in a stethoscope is not to eliminate a certain band of sound frequencies, but to alter the slope of the overall response of the basic curve, as shown in Fig. 19.

Heart and breath sounds are normally impure tones, or noises, and are therefore composed of a conglomeration of frequencies of multitudinous intensities. Each component frequency performs a distinct function, in that it imparts a distinguishing quality to the sound. Although a noise may sound high pitched, wave analysis usually shows that it comprises low, as well as high, frequencies, although the higher frequency components predominate. In cases in which the sounds appear to be low pitched, the reverse usually holds true.

To minimize the masking effects that a more intense and lower pitched sound may produce upon one of high pitch, as, for example, in the case of the aortic diastolic murmur of aortic insufficiency, an attenuating effect upon the lower frequency components would be a distinct ad-

vantage. For best results with the Dodge and Frederick system, using sharp cutoff, high-pass filters, the most suitable point of cutoff will depend upon the component frequencies of the sounds. Considerable variations in the relative proportion of high and low frequencies will be found in cases of aortic regurgitation. On the other hand, a filter such as the "Bowles" diaphragm chest piece, which progressively attenuates, but does not eliminate, the lower frequencies, is obviously less critical and more suitable for clinical use. A modified Dodge and Frederick filter system with a continuously adjustable cutoff filter may be employed. Confusion may arise with such a filter because of the difficulty of selecting and reproducing the optimum cutoff setting in routine clinical work.

Campbell filters, or their modifications, may be so designed that they reproduce the frequency response characteristics of open chest pieces of various sizes, as well as of diaphragm types; the lowest pitch chest piece is employed, and the auscultatory equivalents of the highest pitch chest pieces are obtained by inserting the equivalent electrical filter.

The choice between an acoustic filter and an electrical filter, therefore, reduces itself essentially to a question of economics and weight. Obviously, the acoustic filters are less costly and decidedly less massive. Also, the clinician is more familiar with the acoustic filter system.

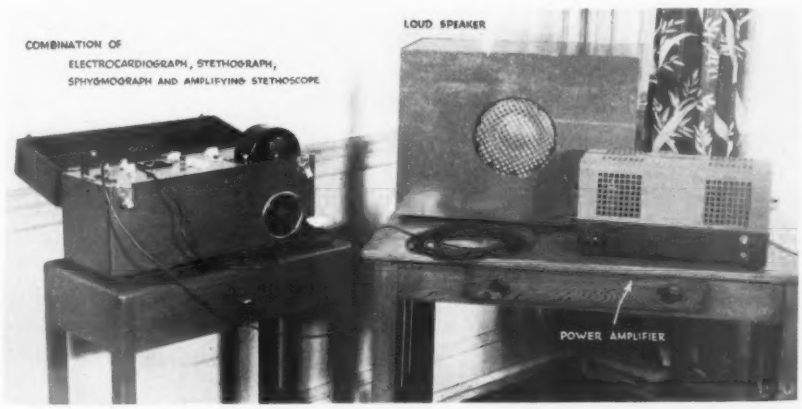

Fig. 20.—Stethograph-electrocardiograph with loud speaker attached.

Applicable Electroacoustic Transducers.—A loud-speaker system designed to operate with the amplifying stethoscope described in this paper is shown in Fig. 20. It is principally useful for teaching, although it has a few clinical applications. Several experimenters have constructed such units, but they have utterly failed to reproduce sounds of unmodified quality. The reasons for the previous failures are as follows:

A. The combinations of microphone and electronic amplification which were used did not possess a frequency response characteristic similar to that of the ordinary acoustic stethoscope.

B. The ordinary radio loud-speakers which were employed did not possess a frequency response characteristic suitable for clinical work.

C. Invariably, the power handling ability of the loud-speakers was insufficient to reproduce the heart sounds with sufficient intensity and with no overloading distortion.

The form of distortion that may be introduced by item A has been discussed previously; it will occur in a loud-speaker system, as well as in an amplifying stethoscope which employs an audiophone.

A loud-speaker which does not possess a frequency response characteristic over the auscultatory range similar to that of the audiophone previously described will alter the quality of the heart sounds. The average radio loud-speaker has a resonance peak in the vicinity of 100 cycles per second, or less. As a result, a booming character is imparted to the first and second heart sounds, producing a tom-tom drum effect. This drum effect, in addition to altering the quality of the heart sounds, decidedly masks any systolic and diastolic murmurs that may be present. The loud-speaker of Fig. 20 is totally free of resonance peaks in the auscultatory region.

We have found that a loud-speaker system must be capable of handling a minimum of 15 watts in order to reproduce the heart sounds without distortion in the average amphitheater or classroom. So much power seems unnecessary, on first thought. However, the large excursions of the loud-speaker diaphragm which are produced by the first and second heart sounds show that a large amount of power is being fed into the loud-speaker. The reason the diaphragm excursions do not produce terrifically loud sounds is that human hearing is very deficient in the frequency band of which these sounds are composed.

In order to deliver sufficient power to the loud-speaker, a power amplifier must be interposed between it and the amplifying stethoscope. The amplifier must not modify the over-all frequency response characteristic of the system.

Clinical tests showed that the loud-speaker system reproduced the heart sounds with a quality that was practically identical with that of the acoustic stethoscope. The efficiency of the acoustic stethoscope was somewhat better at the very low pitches, but the loud-speaker was excellent at the higher pitches. It was also noted that hearing efficiency improved for the very low pitches as the observer came closer to the loud speaker. The reason for this phenomenon is that, at the very low auscultatory frequencies, the sense of feel is more keen than the sense of hearing. A closed binaural conduction system increases the efficiency of feel for the low pitches, as compared to the efficiency of feel with a loud-speaker system.

A suitably designed bone conduction type of transducer may be used in place of the audiophone for the transformation of the amplified

electrical pulsations into sounds. The partially deaf physician whose hearing is more efficient by bone conduction than air conduction will obviously benefit by this arrangement.

The Stethograph.—Earlier in this paper, we described the manner in which the heart sounds are recorded graphically on sensitized photographic paper or film. The same electrical pulsations which pass to the audiophone are applied to the moving-coil galvanometer and are recorded photographically. Thus, an identical and known relationship exists between the sounds as heard and recorded.

The Sphygmograph.—A simultaneously registered stethogram and electrocardiogram, in certain cases, may not supply sufficient information for a complete analysis of all the events of the cardiac cycle. An additional simultaneous recording of a sphygmogram and stethogram, or all three together, must be resorted to. If two of the curves are recorded at the same time, the three may be correlated. Orías and Braun-Menéndez,[71] who have done a considerable amount of work on the physiologic aspects and interpretation of stethograms, state:

"Investigators who persist in the belief that the electrocardiogram will give all the data necessary for the interpretation of phonocardiograms will find themselves seriously limited in their studies, and will not be in a position to make the fullest use of their results."

The authors give as an illustrative example:

"Those workers who have relied on the electrocardiogram alone to localize and correlate the vibrations recorded in the phonocardiogram have been forced to make suppositions based on time intervals between the events in both records. For example, if a sound is produced 0.08 to 0.14 sec. after the beginning of the P wave, it can be assumed that it is connected with auricular contraction; but if the sound is produced in diastole before, or at the same time as, the P wave, all that can be said is that this sound is not connected with auricular contraction. But in which definite moment of diastole is it produced? What is its probable cause? These questions cannot be answered."

Ohm,[72, 73] Groedel,[74] Weber,[75] and Wolferth and Margolis[97-99] also recommend the addition of the phlebogram as a reference tracing.

In general, a tracing of the venous pulse is most useful in the study of the stethogram, for the following reasons:

A. It may be recorded without any undue effort.

B. The delay in the venous pulse when it is recorded at the lower portion of the neck is short and very nearly constant.

C. The venous pulse portrays with good definition the various phases of the cardiac cycle and is an expression of the mechanical activity of the heart which produces the heart sounds.

Although it has been known for several centuries that there are venous pulses, they were not satisfactorily recorded until 1866, by Friedreich.[76] In 1893, Mackenzie,[77, 78] stimulated by a curiosity concerning the possible clinical significance of phlebograms, launched

a series of investigations which produced many enlightening physiologic facts.

The early sphygmographs employed purely mechanical recording systems, with necessarily massive levers and large friction styluses and pens. Wiggers,[24a] in his discussion of the mechanical recording type of polygraph, states:

"In order to render these systems sufficiently sensitive a considerable magnification of the lever is always necessary. In writing as they do, on horizontal surfaces, considerable friction between the writing point and the lever is, moreover, introduced. When, in addition, and contrary to all principles of good lever construction, a considerable weight is placed at the tip in order to enable these levers to record with ink, it can only follow that the instruments are inaccurate: (a) because they have a very slow inherent frequency; (b) because the curves are modified by friction on the drum; and (c) because, being periodic, they introduce lever vibrations which are frequently interpreted as waves of the venous pulse."

Dr. Wiggers obtained the following figures on some of the commonly used sphygmographs:

"Tambours of the Mackenzie ink polygraph have an inherent frequency of 4.5 per second, that of the Zimmermann polygraph, 5.8 per second, while the Jaquet polygraph heads the list with a vibration frequency of 6.5 per second."

In recent years, sphygmographic recording capsules which operate on optical and photosensitive principles have superseded the more sluggish mechanical systems. The Frank[79-82] segment capsule, which employs a beam of light produced by a suitable optical system, instead of a massive mechanical lever and stylus or pen, has been found most satisfactory and has been widely used. The beam of light has no weight, and therefore its movements are not modified or impeded by inertia and friction. Almost any desired degree of optical magnification of the pulse is obtainable, although the magnification is more or less limited by the intensity of the source of light and the necessity of obtaining good photographic definition.

The limiting characteristics of the Frank capsule sphygmograph are as follows:

A. The membrane is a thin rubber dam, to which a small, trapezoidal, celluloid plate is cemented. On the celluloid, in turn, is cemented a tiny mirror which pivots when the rubber diaphragm is excited. The capsule depends upon air damping, and thus a capsule of only a certain natural period will be critically damped. A capsule with a higher natural period will allow overshooting, and superimpose extraneous vibrations upon the sphygmogram, whereas one with a lower natural period will be overdamped and sluggish. As a result, only a critically damped capsule can be used when accurate sphygmograms are desired. It is not a very simple matter to produce critically damped capsules in commercial quantities.

B. The sensitivity of the Frank capsule type of sphygmograph may be controlled by either modifying the length of throw of the recording light beam or substituting capsules with celluloid plates of different trapezoidal shapes. When the optical throw is varied, the sensitivity is increased or decreased in a direct ratio. An elaborate laboratory setup, with an adjustable camera, permits modifications of the optical throw, but this is rather awkward in clinical work. Alterations in the length of the optical beam change the exposure intensity on the photographic paper and the width and photographic sharpness of the recording beam. Substituting capsules with celluloid plates of various trapezoidal shapes in order to obtain the necessary sensitivity is not very practical clinically, for such substitutions take time and require delicate adjustments of the optical system.

C. The life of the Frank capsule is short because the thin rubber dam deteriorates.

In order to overcome the imperfections of the mechanical and optical capsule types of sphygmographs, a piezoelectric system was devised to operate with the stethograph-electrocardiograph. Fig. 21 is a photograph of the sphygmographic unit,* which, when substituted for the patient in the electrocardiographic section of the stethograph-electrocardiograph, will record the sphygmogram.

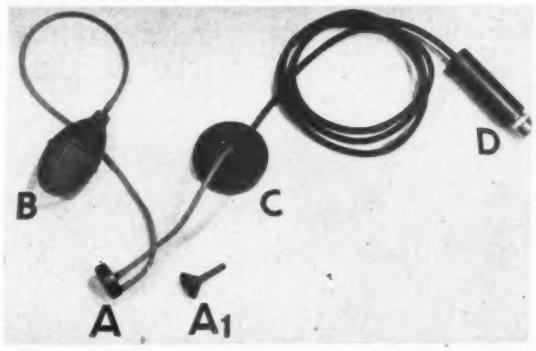

Fig. 21.—Piezoelectric sphygmographic attachment. *A*, Vacuum type applicator cup; A_1, pressure type applicator cup; *B*, rubber bulb to produce vacuum in suction compartment of vacuum type applicator cup; *C*, piezoelectric microphone; *D*, cord and removable connector which fits into patient-cable receptacle of the electrocardiographic section of the instrument.

The attachment sphygmographic unit is composed of *A*, a vacuum type of applicator cup; A_1, an alternate pressure type of applicator cup; *B*, a rubber bulb to produce a vacuum in the suction compartment of the vacuum applicator cup; *C*, a piezoelectric transducer; *D*, a cord and removable connector which fits into a patient-cable receptacle located on the control panel of the stethograph-electrocardiograph.

The vacuum applicator cup consists of a double chamber. The inner chamber is used for picking up the arterial or venous pulsations, whereas the outer chamber, which is connected to bulb *B* through rubber tubing, serves as a suction chamber for fastening the cup to the patient. A pressure type of funnel-shaped applicator (A_1) may be substituted when desired.

The pulse sets up air pressure changes in the applicator cup. The pressure changes are then transmitted through the rubber tubing to the piezoelectric transducer, which converts them into equivalent, minute, electrical pulsations which are, in turn, fed into the electrocardiograph amplifier and recorded like an electrocardiogram.

The piezoelectric transducer operates on a principle identical with that of the stethograph microphone and has a flat frequency response from practically zero to several hundred cycles per second.

The connector *D* is so wired that, when the electrocardiograph selector switch is set in the "Lead I" position, the minute electrical pulsations produced by the piezoelectric transducer are intensified by the electrocardiograph amplifier and recorded photographically. The amplitude of the sphygmographic deflection is regulated by means of the electrocardiograph sensitivity control, and the recording beam is centered by means of the electrocardiograph beam-centering mechanism.

The following are some of the more important characteristics of a piezoelectric sphygmograph when it is used in conjunction with the electronic electrocardiograph:

*This instrument is to be described in a paper by Arthur Miller, Sc.D., and Paul D. White, M.D.

A. The speed of response is limited by the d'Arsonval galvanometer. The galvanometer employed in the electrocardiograph has a deflection time of approximately 0.01 second, as may be seen from the "straight line" graph in Fig. 22. The

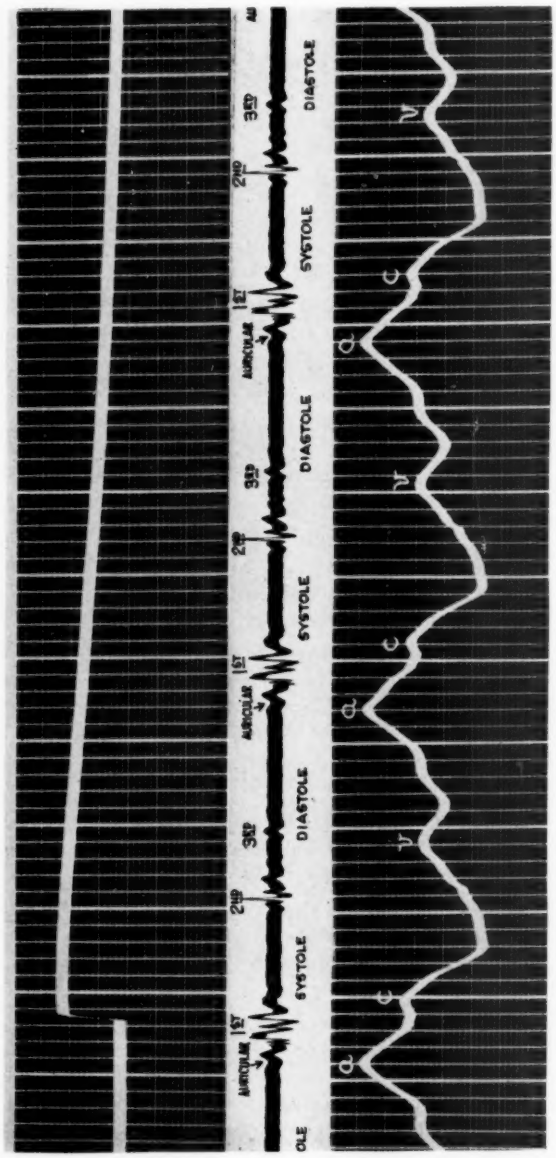

Fig. 22.—Response characteristic of electrocardiograph section.

Fig. 23.—Simultaneously recorded stethogram and venous sphygmogram of a normal person.

"straight line" curve was obtained by applying a potential of 1 millivolt to the input of the amplifier. Each interval between adjacent vertical time lines is equal to exactly 0.04 second. A deflection time of 0.01 second is more than ample for sphygmographic and electrocardiographic purposes.

B. The logarithmic decay characteristic of the amplifier system is also illustrated by Fig. 22. For more than 0.2 second there is no decay whatsoever, and thereafter the decay is gradual. The 0.2 second during which there is no decay is certainly longer than the duration of any known electrocardiographic complex, and, inasmuch as the decay is slow after the 0.2 second, is ample for sphygmographic recording.

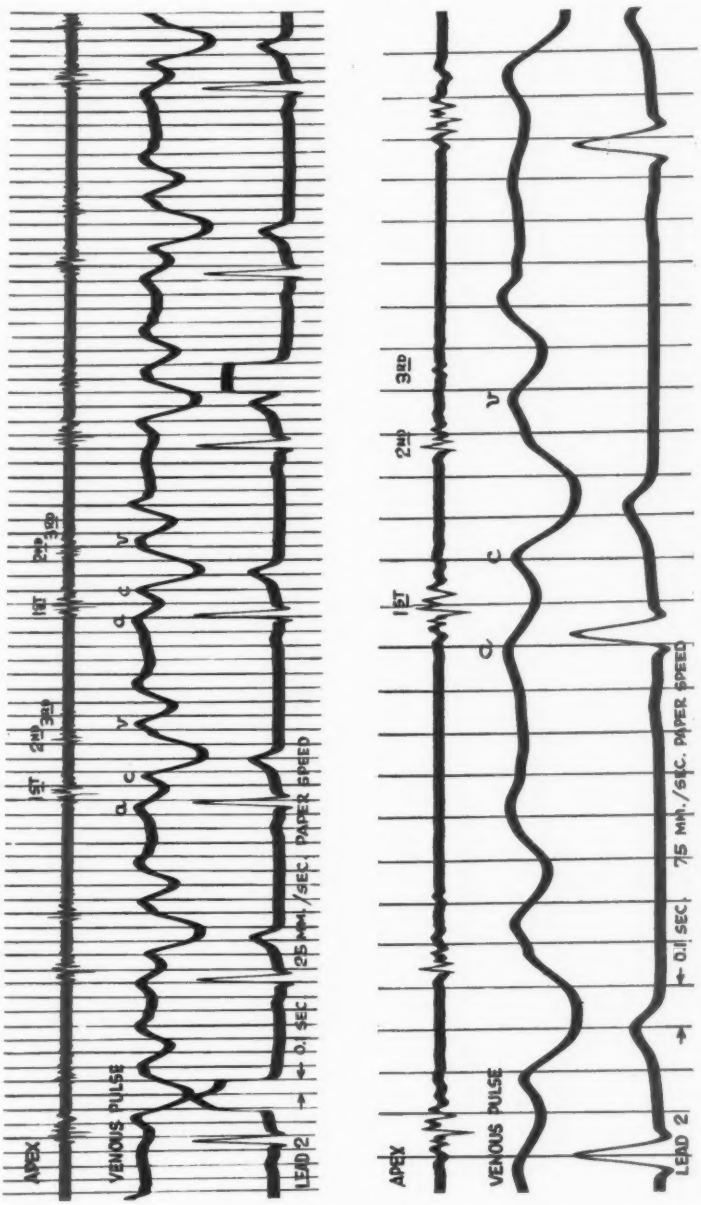

Fig. 24.—Simultaneously recorded stethogram (apex), venous sphygmogram, and electrocardiogram (Lead 2), at 25 and 75 millimeters per second camera speeds.

C. The electrical sensitivity control of the electrocardiograph is simple to use, and it does not alter any characteristic other than the sensitivity of the apparatus.

D. Once the recording beam is set, slight changes in the adjustment of the apparatus will not result in loss of the beam, for it gradually drifts into position because of the decay characteristic of the amplifier.

E. In the circuit there are no delicate parts which require protection.

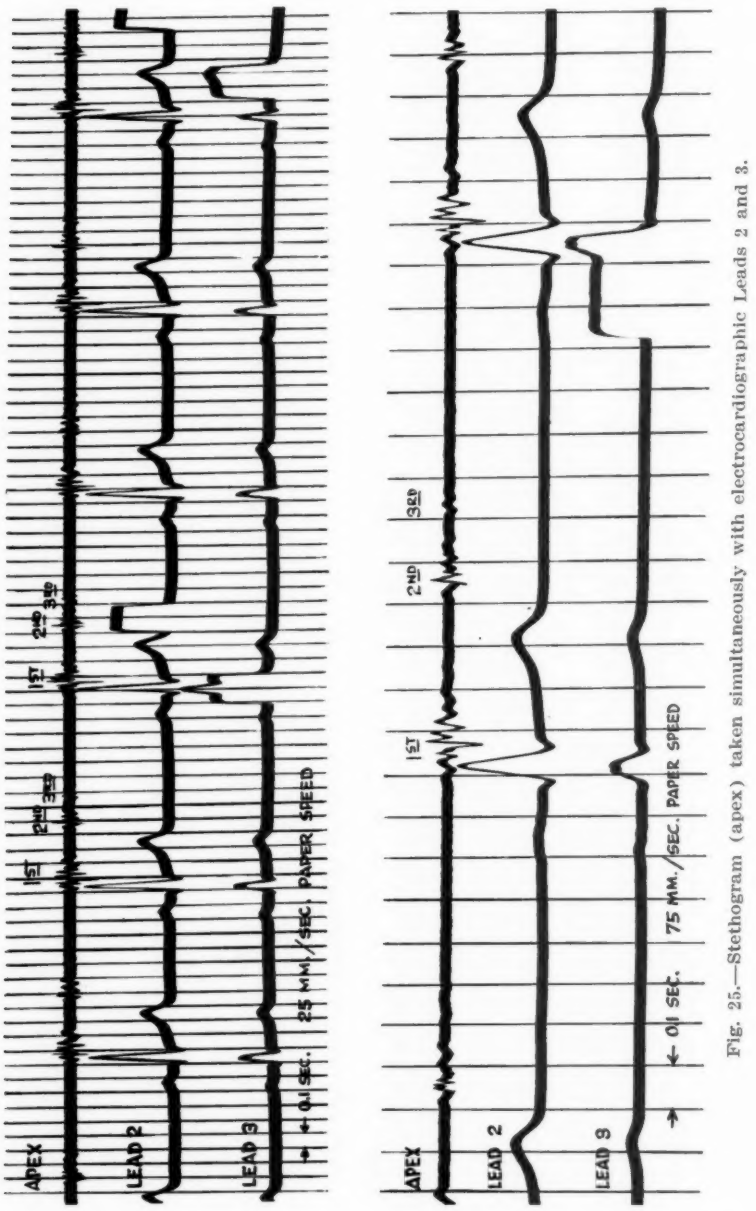

Fig. 25.—Stethogram (apex) taken simultaneously with electrocardiographic Leads 2 and 3.

Fig. 23 shows a venous pulse tracing which was taken simultaneously with the stethogram on a normal subject.

The addition of another electrocardiographic section to the stethograph-electrocardiograph permits registration of three simultaneous physiologic events. The

following combinations are thus made possible: (1) Stethogram, sphygmogram, and one electrocardiographic lead; (2) Stethogram and any two sphygmograms; (3) Stethogram and any two electrocardiographic leads.

When more than two registrations are made simultaneously, it is advisable to employ a white background and record in black, for this allows the three beams to cross each other. Fig. 24 shows a simultaneously recorded stethogram, venous sphygmogram, and electrocardiogram. Fig. 25 illustrates a simultaneously recorded stethogram and two electrocardiographic leads.

VII. Summary and Conclusions

1. Tones of different periods of oscillation or frequency, but of similar intensity, affect the human ear to different degrees. The audiogram, which is a graphic representation of the threshold of audibility, is a measurement of the degree to which human hearing varies with respect to the frequency of vibration of the stimulus.

2. The minimum change in intensity of a sound stimulus to which the human ear is capable of responding varies with the general level of the sound, as well as with its frequency. In the auscultatory frequency band, as the frequency of the stimulus is lowered, a decidedly greater percentage variation in intensity is therefore required to produce the minimum perceptible change.

3. The human ear is a better detector of changes in frequency than of changes in intensity. A sound stimulus with a high sensation level requires less of a frequency variation to produce minimum susceptibility than does a sound stimulus of a lower sensation level. Also, the ear is somewhat less sensitive to frequency variations at the lower end of the auscultatory frequency band than it is to variations in the upper region.

4. In the auscultatory frequency band, the frequency of a stimulus may be varied rapidly over a considerable portion of an octave without detection by the ear.

5. The auditory sensation produced by a complex sound may be decidedly different in character, as well as in intensity, when the stimulating level is decreased or increased, even though no distortion is introduced. As a complex sound, such as a murmur, becomes more intense, the low-pitched components appear more prominent to the observer.

6. When a sound of comparatively high intensity immediately precedes a sound of considerably lower intensity, masking of the sound of lower intensity may result.

7. There are many paths along which heart and chest sounds travel in the human body in order to reach the surface. As a result, a large percentage of the sound energy never reaches the surface because of viscosity, elasticity, density, spreading, reflection, and refraction losses.

8. The entire auscultatory frequency band for heart sounds and murmurs lies below 1,000 cycles per second. An estimation of the lower frequency limit of heart sounds and murmur components puts it in the

vicinity of 5 to 10 cycles per second, although 30 to 40 cycles per second is the lower limit of audibility.

9. Acoustic stethoscopes may be classified as either monaural, binaural, or differential. Either the monaural or binaural stethoscope may be employed for general auscultatory purposes, whereas the differential stethoscopes are primarily instruments for localizing and comparing sounds.

10. The open stethoscopic chest piece, or bell, when applied to the patient's chest, may be considered as a diaphragm type of chest piece. The skin which is bounded by the lip of the bell forms the diaphragm, and the fleshy portion under the skin acts as a damping medium.

11. The larger the diameter of the open stethoscopic chest piece, the better its response to low-pitched sounds. This is accomplished at the expense of the higher frequency components.

12. The greater the pressure with which the open stethoscopic chest piece is applied to the patient's chest, the better is the response of the stethoscope to higher frequency components. Thus, by varying the application pressure, the physician exerts a variable filtering action upon the sounds because the natural period of the skin diaphragm bounded by the chest piece depends on the application pressure.

13. Open stethoscopic chest pieces of various geometrical shapes have been devised to improve the sound-accumulating efficiency of the stethoscope. A bell with its interior shaped like a parabola has been a favorite. Such chest pieces invariably decrease the efficiency of the stethoscope because they increase the internal volume of the chest piece.

14. The only important consideration when designing an open stethoscopic chest piece is to keep its internal volume at a minimum and have it so shaped that, in the case of an obese patient or one with an inelastic chest wall, the bell will not fill with flesh to such an extent as to decrease effectively the diameter of the enclosed diaphragm.

15. The diaphragm type of chest piece (Bowles type), which is commonly used in auscultation, is especially useful in detecting faint, high-pitched sounds. When it is applied to a patient's chest, the principle of operation of the Bowles chest piece is similar to that of the open bell, except that additional attenuation of the lower pitched heart and chest sound components is obtainable with the Bowles chest piece, and this prevents masking of the higher pitched components.

16. In the Bowles chest piece, as in the open type of chest piece, the air volume should be made as small as possible in order to obtain maximum efficiency.

17. Between 60 and 400 cycles per second, which includes most of the auscultatory region, tests show that the binaural method of auscultation through rubber tubes is, on an average, 20 decibels better than the monaural method, with the ear directly applied to the stethoscope. A 20-decibel difference is equivalent to a tenfold increase in sound pressure at the ear drum. Only between 850 and 1,000 cycles per second is

monaural, or direct, auscultation more efficient than binaural, and this range is too high to be practically useful.

18. The changes in the efficiency of an acoustic stethoscope which are caused by varying the length of the tubing, although they are not given any consideration by stethoscope users, produce an effect upon the quality of sounds. Tests show that, below 100 cycles per second, the efficiency is not materially affected by tubing length. Between 100 and 1,000 cycles per second, tubing length exerts a considerable effect; that is, the efficiency decreases with increased tubing length. This efficiency loss occurs in the region of the low intensity, high-pitched, diastolic murmurs, and every possible increase in efficiency in this region is of utmost value.

19. In order to obtain the most efficient tubing dimensions, one should make the tubing as short as possible and compromise on the resistance and volume components. The compromise may be approached by plotting a graph representing efficiency versus volume effect, and another representing efficiency versus frictional resistance effect; where the two curves intersect is the point of optimum efficiency.

20. For general clinical use, an electrical amplifying stethoscope must transmit sounds to the observer with a quality and fidelity equal to that of the average acoustic stethoscope. A modification of the frequency response characteristic of an electrical stethoscope will definitely alter the quality and character of the sounds.

21. An amplifying stethoscope is not primarily an instrument to be used for making sounds many times louder than they can be heard with an acoustic stethoscope. The major advantage of the amplifying stethoscope over the acoustic stethoscope is that it enables one to adjust the intensity to the desired level, and thus eliminate a number of modifying characteristics peculiar to human hearing which cannot be overcome with the acoustic stethoscope.

22. When filters, either electrical or acoustic, are used with an amplifying stethoscope, they should possess frequency response characteristics similar to those of the various open and diaphragm chest pieces.

23. For teaching purposes, a loud-speaker may be used in conjunction with the amplifying stethoscope. The over-all frequency response of the loud-speaker and amplifying stethoscope must be identical with that of the average acoustic stethoscope, in order not to modify the quality and character of the sounds.

24. In order to maintain an identical and known relationship between sounds as heard and as recorded, the recording galvanometer and audiophone must be fed from the same source; that is, the same electrical pulsations which pass to the audiophone are fed into the galvanometer.

25. Making an electrocardiogram simultaneously with the stethogram does not supply enough information for a complete analysis of all the

events of the cardiac cycle, particularly those which occur in diastole. For accurate analysis in such cases, a sphygmographic recording mechanism must be added to the apparatus, so that a simultaneous sphygmogram and stethogrom may be taken to correlate with the simultaneous electrocardiogram and stethogram. A three-element recording system, capable of simultaneously recording the electrocardiogram, stethogram, and sphygmogram, eliminates the correlation procedure.

ADDENDUM

Since the completion of this paper, Johnston and Kline[100] have presented their data on the acoustic stethoscope. The testing procedure employed by these authors is significant, in that it is one of the first attempts at a quantitative analysis of stethoscope performance. A comparison of the data obtained by Johnston and Kline with our observations is of interest.

The experimental setup of Johnston and Kline was composed of: "A special telephone receiver, driven by a variable frequency oscillator, placed within the heart of a cadaver by means of an abdominal-diaphragmatic approach, so that vibrations of any desired amplitude and pitch were transmitted to the intact thoracic wall over the heart. The vibrations were picked up by the end pieces of the stethoscope and conveyed through the columns of air of the instrument to a condenser microphone. The output from the microphone was passed into a suitable vacuum-tube amplifier, and thence to an output meter of the rectifier type. The readings of this meter were proportional to the intensity of the sound waves striking the diaphragm of the condenser microphone. All tests were made with the end pieces of the stethoscope held rigidly against the same point on the precordium, and great care was taken to be sure that each end piece was in air-tight contact with the skin."

The Johnston and Kline calibration procedure was as follows: "Before any tests on stethoscope units were undertaken, attempts were made to standardize the vibrations transmitted to the wall of the chest, so that their amplitude would be constant over the entire range of frequencies employed. This problem proved to be difficult and was not solved to our satisfaction. We finally used a crystal vibration pick-up, fixed at the chosen point on the precordium, and determined the value of the resistance which would give the same response on the output meter at each frequency."

It is our opinion that the Johnston and Kline procedure for obtaining the over-all frequency response characteristic of an acoustic stethoscope gives only a very rough approximation, for two major reasons:

1. A stethoscope does not have similar frequency response characteristics when used on a cadaver and on a living person. The difference in frequency response characteristic is caused by a definite difference

in the diaphragm effect of living and dead skin. Also, the diaphragm damping effect of dead tissue below the dead skin is different from that of living skin and tissue.

2. Unless the volume of the condenser microphone chamber is identical with that of the average human ear, a modification of the response characteristic results. It is hardly conceivable that a condenser microphone could be so constructed.

Johnston and Kline assert that the interposition of a soft rubber nipple between the precordium and the open type of stethoscopic chest piece improves the performance. They state: "It will be observed that, except at the higher frequencies, the use of the nipple results in a significant gain in the response of the unit." Johnston and Kline admit that they cannot explain this phenomenon.

The rubber nipple effect can be explained by means of the skin diaphragm theory. When a soft rubber nipple is interposed between the precordium and the chest piece, more pressure is required to maintain the same natural period of skin diaphragm because of the elasticity of the rubber. Also, to obtain a good seal between the lip of the bell and the skin, less pressure is required when soft rubber is interposed.

Thus, the conclusion drawn by Johnston and Kline requires modification, in that the interposition of a soft rubber nipple reduces the natural period of the skin diaphragm, with the following effects:

1. The low-pitched sounds are increased in intensity, whereas the high-pitched sounds are decreased; this was observed by Johnston and Kline and is a normal diaphragm characteristic.

2. Because of item 1, the pitch of the sounds is made lower.

3. The interposition of a soft rubber nipple is equivalent to employing an open chest piece of larger diameter.

Johnston and Kline show that an open chest piece with a large, conical air chamber in the bell compares favorably with a shallow one up to 100 cycles per second, but becomes less efficient above 100 cycles per second. Our observations are in agreement with these.

Another observation by Johnston and Kline is as follows: "A soft rubber sheath placed over the aperture of a large Bowles unit materially improves its transmission, and this is true whether a diaphragm is used or not." When a Bowles chest piece is used in auscultation, the quality and intensity of the heart sounds depend considerably on whether the pressure is light or moderately heavy. With light pressure, the pitch of the sounds is lower and the intensity greater, and with moderately heavy pressure the pitch is considerably higher and the intensity less. Obviously, the interposition of a soft rubber sheath produces the effect of less pressure, and thus lowers the pitch and increases the intensity. The damping effect on the Bowles diaphragm of an interposed rubber sheath is different from that of skin and tissue. In other words, the interposition of the rubber sheath alters the frequency re-

sponse characteristic of the stethoscope and defeats, to a certain extent, the purpose of the Bowles diaphragm. The removal of the Bowles diaphragm produces a still further modification, in that it becomes a hybrid open chest piece.

Johnston and Kline conclude that: "Although rubber tubing of different lengths, diameters, and degrees of stiffness modifies transmission, largely because of resonance phenomena, the differences are relatively small, and the response of a stethoscope depends more on the choice of the end piece than on the nature of the tubes that are used." Our data differ considerably on these points.

Above about 150 cycles per second, tubing length is a very important factor in stethoscopic efficiency. According to our Fig. 9, there is a mean difference between the energy levels of 3-inch and 26-inch tubes of approximately 10 decibels, which is a pressure ratio of about 3 to 1, as applied to the ears. This means that above approximately 150 cycles per second the average hearing ability with 3-inch stethoscope tubes is roughly four times as great as with 26-inch tubes—certainly a significant amount!

Furthermore, tubing length does not materially modify the quality of sounds, whereas the diameter of the chest piece or Bowles diaphragm does affect the quality. Any variations in the stethoscope frequency response below about 200 cycles per second result in a decided alteration in sound quality; above 200 cycles per second very little alteration takes place. Stethoscope tubing produces little change in the frequency response curve below 200 cycles per second, whereas the chest piece modifies the slope considerably. Therefore, it is not correct to speak of efficiency versus tubing length and chest piece, unless the frequency response and its effects upon human hearing are taken into account; this Johnston and Kline have not done.

The effects of stethoscope resonance on efficiency are small compared to tubing length because the major resonance peaks do not exceed the mean by more than about 5 decibels, and the individual peaks cover but a small portion of the auscultatory band. Also, the location of the resonance peaks may vary as a result of differences in observer's ear cavity volume, leakage, variations in commercial tubing, etc.

In order to increase the efficiency of the stethoscope, Johnston and Kline advise a reduction in the diameter of the tubing. They do not consider the fact that there is an optimum diameter for maximum efficiency. When the diameter is decreased beyond a certain point, losses caused by frictional resistance offered to the air column exceed the gains obtained by volume reduction.

Johnston and Kline finally conclude as follows: "We do not advise all physicians to discard the stethoscope to which they are accustomed for one that may be acoustically superior, because, except for sounds

that are so faint as to be nearly inaudible, the loudness of sounds through the instrument is of no importance.'' This statement is true if we disregard the characteristics of human hearing, as Johnston and Kline have done, but, if we do consider these characteristics, it is not true. We have pointed out that the degree of masking, as well as the quality of the sounds, may be affected by the loudness level.

REFERENCES

1. Forbes, John: The Stethoscope and Percussion in the Diagnosis of Diseases of the Chest; also Commentaries on the Same Subjects Selected and Translated from Auenbrugger, Corvisart, Laennec, and Others, Printed for T. and G. Underwood, Fleet Street, London, 1824.
2. Fletcher, Harvey: Speech and Hearing, New York, 1929, D. Van Nostrand Co., Inc.
3. Toepler and Boltzmann: Ueber eine neue optische Methode, die Schwingungen tönender Luftsäulen zu analysiren, Ann. d. Physik u. Chemie **141**: 321, 1870.
4. Rayleigh, Lord: On the Amplitude of Sound Waves, Proc. Roy. Soc. London **26**: 248, 1877.
5. Wead, C. K.: On the Intensity of Sound, Am. J. Sc. **26**: 177, 1883.
6. Wein, Max: Ueber die Empfindlichkeit des menschlichen Ohres für Töne verschiedener Höhe, Arch. f. d. ges. Physiol. **97**: 1, 1903.
7. Boltzmann, F. L.: Festschrift, Leipzig, 1904, Barth, p. 1866.
8. Abraham, Henri: Sensibilité absolue de l'oreille, Compt. Rend. Acad. d. sc. **144**: 1099, 1907.
9. Kranz, F. W.: Minimum Intensity of Audition, Physical Rev. **21**: 573, 1923.
10. Wegel, R. L.: The Physical Examination of Hearing and Binaural Aids for the Deaf, Proc. Nat. Acad. Sc. **8**: 155, 1922.
11. Knudsen, V. O.: The Sensibility of the Ear to Small Differences in Intensity and Frequency, Physical Rev. **21**: 84, 1923.
12. Shower, E. G., and Biddulph, R.: Differential Pitch Sensitivity of the Ear, J. Acous. Soc. America **3**: 275, 1931.
13. Stewart, G. W.: Problems Suggested by an Uncertainty Principle in Acoustics, J. Acous. Soc. America **3**: 325, 1931.
14. Mayer, A. A.: Researches in acoustics, Phil. Mag. & J. Sc. **2**: 500, 1876.
15. Kingsbury, B. A.: A Direct Comparison of the Loudness of Pure Tones, Physical Rev. **29**: 588, 1927.
16. Fletcher, H., and Munson, W. A.: Loudness, Its Definition, Measurement, and Calculation, J. Acous. Soc. America **5**: 82, 1933.
17. Trendelenburg, F.: Physical and Physiological Problems in Examination and Interpretation of Heart Sounds, Med. Klin. **28**: 146, 1932.
18. Williams, H. B., and Dodge, H. F.: Analysis of Heart Sounds, Arch. Int. Med. **38**: 685, 1926.
19. Gamble, C. J., and Replogle, D. E.: A Multiple Electrical Stethoscope for Teaching, J. A. M. A. **82**: 387, 1924.
20. Pierach, Alex: Studien über klinische Akustik, Deutsches Arch. f. klin. Med. **171**: 234, 1931.
21. Landes, G.: Weitere Untersuchungen über die Schallübertragung des Stethoskops, Deutsches Arch. f. klin. Med. **171**: 607, 1931.
22. Cabot, R. C., and Dodge, H. F.: Frequency Characteristics of Heart and Lung Sounds, J. A. M. A. **84**: 1793, 1925.
23. Stevens, S. S., and Davis, H.: Hearing—Its Psychology and Physiology, New York, 1938, John Wiley & Sons, Inc.

24a. Wiggers, C. J.: Circulation in Health and Disease, Philadelphia and New York, 1923, Lea & Febiger, p. 222.

24b. Ibid., ed. 2, 1937, p. 581.

25. de Forest, Lee: U. S. Patent 879,532, Filed Jan. 29, 1907; British Patent 1,427, Filed Jan. 21, 1908.
26. Dodge, H. F.: The Stethophone, Electrical Communication **3**: 106, 1924.
27. Frederick, H. A., and Dodge, H. F.: The Stethophone, An Electrical Stethoscope, Bell Telephone Laboratories, Reprint B-104-1, Jan., 1925.
28. Battaerd, P. J. T. A.: Further Graphic Researches on the Acoustic Phenomena of the Heart in Normal and Pathological Conditions, Heart **6**: 121, 1915.

29. Brömser, P., and Frank, O.: Ein neues Verfahren zur Registrierung von Schallphänomenen, Gesellsch. f. Morphol. u. Physiol.—Sitzungsb. **27**: 46, 1913.
30. Crehore, A. C., and Meara, F. S.: The Micrograph, J. Exper. Med. **13**: 616, 1911.
31. Einthoven, W.: Die Registrierung der menschlichen Herztöne mittels des Saitengalvanometers, Pflüger's Arch. f. d. ges. Physiol. **117**: 147, 1907.
32. Geluk, M. A. J., and Einthoven, W.: Registration by Capillary Electrometer, Arch. f. d. ges. Physiol. **57**: 617, 1894.
33. Fahr, George: On Simultaneous Records of the Heart Sounds and the Electrocardiogram, Heart **4**: 147, 1912.
34. Frank, O.: Die unmittelbare Registrierung der Herztöne, München. med. Wchnschr. **51**: 953, 1904.
35. Frank, O.: Dynamics of Air Transmission, Ztschr. f. Biol. **50**: 341, 1907.
36. Frank, O.: Cardiograms and Heart Sounds, Klin. Wchnschr. **45**: 1150, 1908.
37. Frank, O.: Principles of Sound Registration, Ztschr. f. Biol. **55**: 530, 537, 1911.
38. Frank, O.: Die Prinzipien der Schallregistrierung, Ztschr. f. Biol. **64**: 125, 1914.
39. Gerhartz, H.: Die Aufzeichnung von Schallerscheinungen insbesondere die des Herzschalles, Ztschr. f. exper. Path. u. Therap. **5**: 105, 1908.
40. Hermann, L.: Die theoretischen Grundlagen für die Registrierung akustischer Schwingungen, Arch. f. d. ges. Physiol. **150**: 92, 1913.
41. Lilienstein: Über die akustischen Besonderheiten der Herztöne (Ein neuer Herzkontrollapparat), München med. Wchnschr. **58**: 1561, 1911.
42. Myres, M. J.: The Clinical Application of the Audion Amplifier, J. A. M. A. **78**: 100, 1922.
43. Ohm, R.: Registration by Gelatine Capsule Method, Ztschr. f. exper. Path. u. Therap. **11**: 138, 1912.
44. Wiggers, C. J., and Dean, A. L., Jr.: The Principles and Practice of Registering Heart Sounds by Direct Methods, Am. J. M. Sc. **153**: 666, 1917.
45. Williams, H. B.: A New Method for Graphic Study of Heart Sounds, Proc. Soc. Exper. Biol. & Med. **18**: 179, 1921.
46. Sawyer, C. B.: The Piezo-Electric Crystal, Inst. Radio Engineers Proc. **19**: 2020, 1931.
47. Williams, A. L.: Piezo-Electric Microphone and Bibliography, J. Soc. Motion Picture Eng. **4**: 196, 1934.
48. Wright, C. A., and Puchstein, A. F.: Telephone Communication, New York, 1925, McGraw-Hill Book Co.
49. Gregory, H. J.: The Carbon Transmitter, Post Office Elec. Eng. J. July, 1920.
50. Jones, W. C.: Condenser and Carbon Microphones—Their Construction and Use, Bell System Tech. J. **10**: 46. 1931.
51. Crandall, I. B.: The Condenser Microphone, Physical Rev. **11**: 449, 1918.
52. Hall, W. M.: Condenser Microphone, J. A. S. A. **4**: 83, 1932.
53. Harrison, H. C., and Flanders, P. B.: Condenser Microphone, Bell System Tech. J. **11**: 451, 1932.
54. Oliver, D. A.: An Improved Condenser Microphone for Sound Pressure Measurements, J. Scient. Instruments **7**: 113, 1930.
55. Wente, E. C.: Condenser Microphone, Physical Rev. **10**: 39, 1917; also **19**: 498, 1922.
56. West, W.: Condenser Microphones, J. I. E. E. **67**: 1137, 1929; also **68**: 145, 1930.
57. Campbell, G. A.: Electric Wave Filters, Bell System Tech. J. **1**: 1, 1922.
58. Stewart, G. W.: Acoustic Wave Filters, Physical Rev. **20**: 528, 1922.
59. Zobel, O. J.: Theory and Design of Uniform and Composite Electric Wave-Filters, Bell System Tech. J. **2**: **1**, 1923.
60. Johnson, K. S.: Transmission Circuits for Telephonic Communication, New York, 1925, D. Van Nostrand Co., Inc.
61. Shea, T. E.: Transmission Networks and Wave Filters, New York, 1929, D. Van Nostrand Co., Inc.
62. Mason, W. P.: A Study of the Regular Combination of Acoustic Elements, With Applications to Recurrent Acoustic Filters, Tapered Acoustic Filters and Horns, Bell System Tech. J. **6**: 258, 1927.

63. Crandall, I. B.: Theory of Vibrating Systems and Sound, New York, 1926, D. Van Nostrand Co., Inc.
64. Olson, H. F., and Massa, F.: Applied Acoustics, Philadelphia, 1939, P. Blakiston's Son & Co.
65. Pender, H., and McIlwain, K.: Electrical Engineers Handbook, Vol. Electric Communication and Electronics, New York, 1936, John Wiley & Sons, Inc.
66. Kennelly, A. E.: Electrical Vibration Instruments, New York, 1923, The Macmillan Co.
67. Lamb, H.: The Dynamical Theory of Sound, London, 1910, Edward Arnold & Co.
68. Chaffee, E. L.: Theory of Thermionic Vacuum Tubes, New York, 1933, McGraw-Hill Book Co.
69. Rich, G. J.: A Preliminary Study of Tonal Volume, J. Exper. Psychol. **1**: 13, 1916.
70. Dodge, H. F., and Frederick, H. A.: U. S. Patent 1,686,504, Filed May 29, 1924.
71. Orías, O., and Braun-Menéndez, E.: The Heart Sounds in Normal and Pathological Conditions, London, New York, and Toronto, 1939, Oxford University Press.
72. Ohm, R.: Venenpuls und Herztöne, Deutsche med. Wchnschr. **40**: 493, 1914.
73. Ohm, R.: Venenpuls und Herzschallregistrierung als Grundlage für die Beurteilung der mechanischen Arbeitsleistung des Herzens nach eigenen Methoden, Berlin, 1914, A. Hirschwald.
74. Groedel, F. M.: Die diagnostische Bedeutung der graphischer Darstellung der Herztöne, VI, Fortbild. Lehr. in Bad Nauheim, 18-ix-1929, Leipzig, 1930, Georg Thieme.
75. Weber, A.: Gleichzeitige Aufnahme von Elektrokardiogramm, Herztönen, und Venenpuls, Klin. Wchnschr. **10**: 575, 1931.
76. Friedreich, N.: Über den Venenpuls, Deutsches Arch. f. klin. Med. **1**: 241, 1866.
77. Mackenzie, J.: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart, New York and London, 1902, The Macmillan Co.
78. Mackenzie, J.: The Ink Polygraph, Brit. M. J. **1**: 1411, 1908.
79. Frank, O.: Segment Capsule, Theory, Ztschr. f. Biol. **50**: 281, 1908.
80. Frank, O.: Segment Capsule, Theory, Tigerstedt's Handb. der Physiol. Methodik. **112**: 94, 1913.
81. Frank, O.: Technic u. Method (photokymograph), Ztschr. f. Biol. **50**: 303, 1908.
82. Brömser, P., Frank, O., and Petter, J.: Experimentelle Prüfung der Frankschen Theory der Schwingungen von Flüssigkeitssäulen, Ztschr. f. Biol. **50**: 232, 1912.
83. Fröschels, E.: Ein Differenzstethoskop, Med. Klin. **30**: 1099, 1934.
84. Nicolai, L.: Über das "Stereostethoskop.," Klin. Wchnschr. **36**: 91, 1936.
85. Kerr, W. J., Althausen, T. L., Bassett, A. M., and Goldman, M. J.: The Symballophone, Am. Heart J. **14**: 594, 1937.
86. Muralt, L. V.: Zur Kenntnis der symmetrisch fortgeleiteten Rasselgeräusche, Beitr. z. Klin. d. Tuberk. **16**: 121, 1910.
87. Nicolai, L., and Hantschmann, L.: Erfahrungen mit dem "Stereostethoskop" in der inneren Medizin, Klin. Wchnschr. **15**: 92, 1936.
88. Alison, S. Scott: The Employment of Water in Auscultation, M. Times & Gaz. **19**: 7, 28, 1859.
89. Hawthorne, C. O.: Differential (Double) Stethoscope, Irish J. M. Sc. **110**: 49, 1935.
90. Barss, W. R., Eade, W. F., and Fitzgerald, E. B.: Stethoscopes, Boston M. & S. J. **195**: 116, 1926.
91. Tobler, W.: Testing Stethoscopes, Schweiz. med. Wchnschr. **56**: 873, 1926.
92. Sell, H.: Eine neue Apparatur zur Wiedergabe und Aufzeichnung von Auskultationsphänomenen, Mediz. Sekt. der Schlesichen Ges. f. vaterländische Kultur zu Breslau, Sitz. 11. Dez. 1931 in Klin. Wchnschr. **11**: 834, 1932.
93. Sell, H.: Eine neue Methode zur Aufzeichnung und Wiedergabe von Auskultationsphänomenen, Med. Klin. **28**: 150, 1932.
94. Trendelenburg, F.: Über Herztonverstärkung, Ztschr. f. Kreislaufforsch. **20**: 436, 1928.
95. Trendelenburg, F.: Electrische Methoden zur Klanganalyse, in Abderhalden's Handbuch d. biologischen Arbeitsmethoden, Abt. V, T. 7/1, 787, 1930.

96. Trendelenburg, F.: Klänge und Geräusche. Methoden und Ergebnisse der Klangforschung. Schallwahrnehmung. Grundlegende Fragen der Klangübertragung., Berlin, 1935, Julius Springer.
97. Wolferth, C. C., and Margolies, A.: The Influence of Auricular Contraction on the First Heart Sound and the Radial Pulse, Arch. Int. Med. **46**: 1048, 1930.
98. Margolies, A., and Wolferth, C. C.: The Opening Snap in Mitral Stenosis, Its Characteristics, Mechanism of Production, and Diagnostic Importance, AM. HEART J. **7**: 443, 1932.
99. Wolferth, C. C., and Margolies, A.: Asynchronism in Contraction of the Ventricles in the So-Called Common Type of Bundle-Branch Block: Its Bearing on Determination of the Site of the Significant Lesions and on the Mechanism of the Split First and Second Heart Sounds, AM. HEART J. **10**: 425, 1935.
100. Johnston, F. D., and Kline, E. C.: An Acoustical Study of the Stethoscope, Arch. Int. Med. **65**: 328, 1940.

THE EFFECT OF RE-ESTABLISHMENT OF CIRCULATION IN COMPLETELY ISCHEMIC KIDNEYS UPON THE BLOOD PRESSURE OF CATS, DOGS, AND RATS

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IN 1938, Taquini[1] reported that, when the kidney of a dog is made completely ischemic, re-establishment of the renal circulation is followed by a rise in blood pressure. In a previous study,[2] it was found that the expected rise in blood pressure did not occur in rabbits following the termination of complete renal ischemia which had lasted four to six hours. At that time, the only way to account for the difference in the response of dogs and rabbits was to assume a species variation. Because of the negative response in rabbits, it seemed desirable to extend these observations to other animals; in this communication we are reporting the effects upon the blood pressure of re-establishment of the circulation of ischemic kidneys in cats, dogs, and rats.

CATS

Method.—Ninety-five experiments were performed upon eighty-four animals. Under ether anesthesia, the abdomen was opened, and, following removal of the perirenal fat (to eliminate collateral circulation to the cortex) and stripping of the renal pedicle, a bulldog clamp was applied to the entire left or right renal pedicle. The abdominal cavity was closed and the animal was allowed to recover from the anesthetic. Three to eight hours later the cat was anesthetized with nembutal, given intraperitoneally; the carotid was cannulated, and the blood pressure was recorded on a moving drum in the usual manner. The abdomen was then reopened through the previous incision. Following the establishment of a constant blood pressure level, the bulldog clamp was quickly and carefully removed from the renal pedicle. Changes in the blood pressure were observed.

Six of the cats served for two experiments each; the first kidney was removed after the first experiment; the second experiment, utilizing the remaining kidney, usually took place one week after the first. Five other cats also served for two experiments each; bulldog clamps were placed on both renal pedicles of each animal during the first operation. Several hours later one clamp was removed, and, after the change in blood pressure had apparently run its course and the pressure had returned to or near the previous level, the other clamp was released and changes in blood pressure were observed.

After completion of this part of the experiment, the kidney whose pedicle had been clamped was tested to determine whether its circulation had been adequately restored following removal of the clamp. The following tests were employed:

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TABLE I

EFFECT OF RE-ESTABLISHMENT OF THE CIRCULATION OF COMPLETELY ISCHEMIC KIDNEYS OF CATS

ANIMAL NO.	PEDICLE CLAMPED	TIME CLAMPED (HOURS)	B.P. BEFORE REMOVAL OF CLAMP (MM. Hg)	B.P. FOLLOWING REMOVAL OF CLAMP (MM. Hg)	NET RISE IN B.P. (MM. Hg)	STATE OF RENAL CIRCULATION
1	R	5	185	199	14	Good
2	R	6½	160	178	18	Good
3	R	5	192	212	20	Good
4	R	6½	166	166	0	Good
5	R	3	164	174	10	Good
6	R	5	136	162	26	Good
7	L	6	150	136	−14	Good
8	L	4	209	224	13	Good
9	L	4	186	206	20	Good
10	R	4	125	147	22	Good
11	R	4½	178	212	34	Good
12	L	5	142	170	28	Good
13	L	5	166	182	16	Good
14	R	5	162	179	17	Good
15	L	6	160	172	12	Good
16	L	4½	134	156	22	Good
17	R	4	144	148	4	Good
18	R	6	128	182	54	Good
19	L	4½	164	244	80	Good
20	L	4	144	180	36	Good
21	R	4	128	168	40	Good
22	L	4½	162	184	22	Good
23	L	5	140	176	36	Good
24	L	4	144	160	16	Good
25	R	4½	144	190	46	Good
26	L	4	140	114	−26	Good
27	L	5	160	124	−36	Fair
28	L	3	162	190	28	Good
29	R	3	185	199	14	Good
30	L	5	188	200	12	Good
31	L	5	178	220	42	Good
32	L	5	126	116	−10	Good
33	L	7	164	170	6	Good
34	L	5	156	162	6	Good
35	L	7	172	178	6	Good
36	L	4	195	199	4	Good
37	L	7	210	222	12	Good
38	L	8	166	218	52	Good
39	L	7	138	242	104	Good
40	L	5	156	174	18	Good
41	L	5	124	176	52	Good
42	L	5	144	184	40	Good
43	L	5	152	208	56	Good
44	L	5	188	216	28	Good
45	L	5	124	144	20	Good
46	L	5	70	136	66	Good
47	L	5	160	182	22	Good
48	L	3	174	212	38	Good
49	L	5	126	184	58	Good
50	L	5½	140	158	18	Good
51	L	5	140	230	90	Good
52	L	6	170	198	28	Good
53	L	5	148	188	40	Good
54	L	6	186	220	34	Good
55	L	7	156	180	24	Good
56	L	4	180	234	54	Good

TABLE I—CONT'D

ANIMAL NO.	PEDICLE CLAMPED	TIME CLAMPED (HOURS)	B.P. BEFORE REMOVAL OF CLAMP (MM. Hg)	B.P. FOLLOWING REMOVAL OF CLAMP (MM. Hg)	NET RISE IN B.P. (MM. Hg)	STATE OF RENAL CIRCULATION
57	L	4½	154	196	42	Good
58	L	5½	178	192	14	Good
59	L	3½	76	132	56	Good
60	L	4½	186	224	38	Good
61	L	5	202	226	24	Good
62	L	4½	166	192	26	Good
63	L	4	206	214	8	Good
64	L	4	156	172	16	Good
65	L	4	174	260	86	Good
66	L	5½	192	206	14	Good
67	L	4	150	196	46	Good
68	L	5	166	226	60	Good
69	L	5	154	252	98	Good
70	L	4½	186	210	24	Good
71	R	3	182	206	24	Good
72	L	3	192	200	8	Good
73	L	5½	176	200	24	Good
74	L	3½	200	218	18	Good
75	R	5½	156	176	20	Good
76	L	7	144	186	42	Good
77	L	3½	122	148	26	Good
78	L	4½	178	196	18	Good
79	L	4½	172	206	34	Good
80	L	6	180	232	52	Good
81	L	4	172	252	80	Good
82	L	4	188	220	32	Good
83	L	5	176	224	48	Good
84	L	5½	158	180	22	Good
85	L	3½	182	218	36	Good
86	R	4	200	228	28	Good
87	L	4			28	Good
88	R	4			68	Good
89	L	3	108	146	38	Good
90	R	3	140	158	18	Good
91	L	4½	160	170	10	Good
92	R	4½	178	196	18	Good
93	L	5½	200	238	38	Good
94	R	5½	216	254	38	Good
95	R	3½	160	210	50	Good
Av.		5½			30.12	

(1) A dose of 0.025 mg. of epinephrine was injected into the substance of the kidney; an immediate rise in systemic blood pressure indicated that the renal circulation had been re-established.

(2) The cortex of the kidney was punctured with a pointed instrument; profuse bleeding demonstrated that the vessels were patent.

RESULTS

In eighty-three of the ninety-five experiments, re-establishment of the circulation in the completely ischemic kidney resulted in an elevation of the blood pressure of at least 10 mm. Hg; the average was 35 mm. Hg. In seven experiments the rise in blood pressure was less than 10 mm. Hg; in one experiment there was no rise in blood pressure; and in four experiments there was a fall. The average rise in blood pressure for all ninety-five experiments was 30.12 mm. Hg (Fig. 1, Table I).

The blood pressure usually began to rise five to thirty seconds after the removal of the clamp, reached its maximum after two to four minutes, and then gradually returned to its former level. Although no

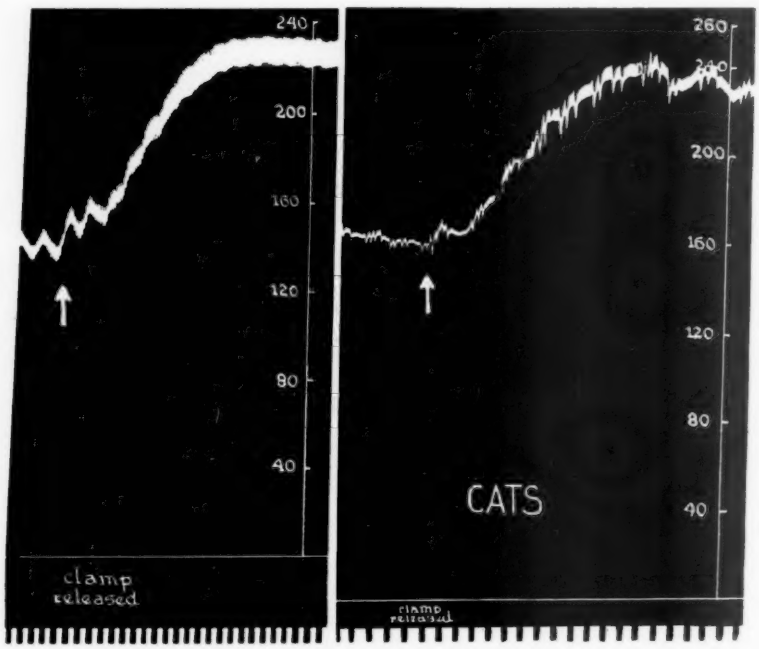

Fig. 1.—Kidneys completely ischemic for five hours. In each instance, clamp was removed at arrow, re-establishing circulation in the ischemic kidney. Nembutal anesthesia. Blood pressure in millimeters Hg. Time marker, fifteen seconds.

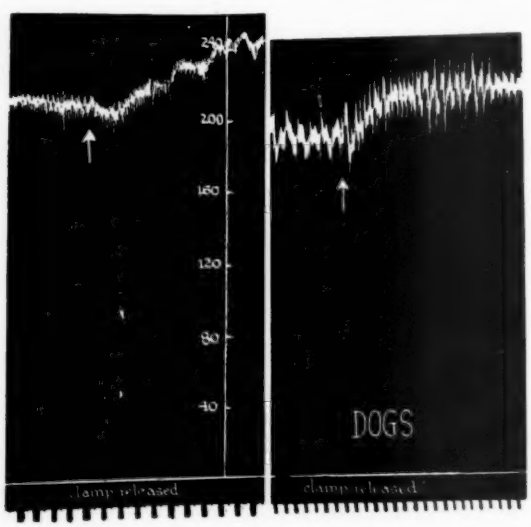

Fig. 2.—Kidneys completely ischemic for five hours. In each instance, clamp was removed at arrow, re-establishing circulation in the ischemic kidney. Nembutal anesthesia. Blood pressure in millimeters Hg. Time marker, fifteen seconds.

effort was made to correlate exactly the duration of the ischemia with the rise in blood pressure, there was no significant difference after varying periods of ischemia. It is likely that if intervals of less than three hours had been employed, a difference would have been apparent.

DOGS

Method.—There were seven animals in this series. The technique was the same as that used on cats, except that all of the dogs were anesthetized by giving them nembutal intraperitoneally for the first as well as the second operation. Renal ischemia was maintained in this series for five to eight hours. The kidney circulation was tested as previously described for cats.

RESULTS

In two of the seven experiments the renal circulation was not re-established, and these were discarded. A significant rise in blood pressure occurred in four of the remaining five animals; the average rise was 21.4 mm. Hg (Fig. 2, Table II).

TABLE II

EFFECT OF RE-ESTABLISHMENT OF THE CIRCULATION OF COMPLETELY ISCHEMIC KIDNEYS OF DOGS

ANIMAL NO.	PEDICLE CLAMPED	TIME CLAMPED (HOURS)	B.P. BEFORE REMOVAL OF CLAMP (MM. Hg)	B.P. FOLLOWING REMOVAL OF CLAMP (MM. Hg)	NET RISE IN B.P. (MM. Hg)	STATE OF RENAL CIRCULATION
1	L	6½	135	155	20	Good
2	L	5½	210	248	38	Good
3	R	5½	172	174	2	Good
4	L	5½	182	199	17	Good
5	R	6	190	220	30	Good
Av.		6	177.8	199.2	21.4	

TABLE III

EFFECT OF RE-ESTABLISHMENT OF THE CIRCULATION OF COMPLETELY ISCHEMIC KIDNEYS OF RATS

ANIMAL NO.	PEDICLE CLAMPED	TIME CLAMPED (HOURS)	B.P. BEFORE REMOVAL OF CLAMP (MM. Hg)	B.P. FOLLOWING REMOVAL OF CLAMP (MM. Hg)	NET RISE IN B.P. (MM. Hg)	STATE OF RENAL CIRCULATION
1	L	5½	136	160	24	Good
2	L	5	141	176	35	Good
3	L	6	144	182	38	Good
4	L	6½	136	215	79	Good
Av.		6	139	183	44	

RATS

Method.—Four animals were anesthetized with ether. In each case the abdomen was opened through a midventral incision, the left kidney and its pedicle were carefully isolated, and a bulldog clamp was placed upon the pedicle. The abdomen was then closed, and the animal was allowed to recover from the anesthetic. Five to six and one-half hours later the rat was reanesthetized with nembutal, given intraperitoneally; the abdomen was reopened through the previous incision; the abdominal

aorta was cannulated with a three-way metal cannula; and the blood pressure was recorded on a moving drum. When a constant blood pressure level had been established, the clamp was removed from the renal pedicle and changes in blood pressure were observed. The state of the circulation in the kidney was tested by injection of epinephrine into the kidney substance and by puncture of the renal cortex, as previously described.

RESULTS

There was a significant rise in blood pressure after removal of the clamp from the renal pedicle in every instance; the average rise was 44 mm. Hg (Fig. 3, Table III).

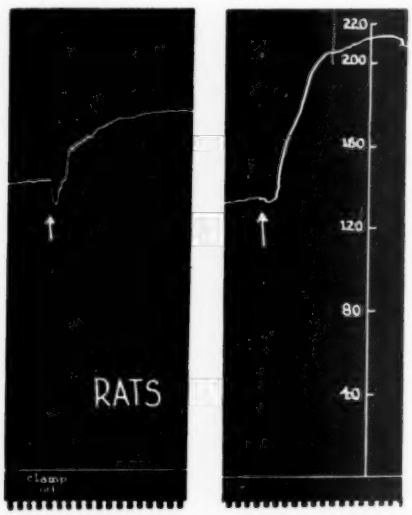

Fig. 3.—Kidneys completely ischemic for five hours. In each instance, clamp was removed at arrow, re-establishing circulation in the ischemic kidney. Nembutal anesthesia. Blood pressure in millimeters Hg. Time marker, fifteen seconds.

DISCUSSION

These observations demonstrate that re-establishment of the circulation in the completely ischemic kidney in cats, dogs, and rats is followed by a rise in blood pressure. In previous communications[3, 4] it was shown that this rise is caused by the release into the general circulation of a pressor substance, probably renin or a closely related substance, which is produced in the ischemic kidney. It was also shown that the kidneys of rabbits which had been rendered completely ischemic for four to six hours fail, for some unexplained reason, to produce the pressor substance. This accounts for the fact that the blood pressure does not rise in rabbits.

SUMMARY AND CONCLUSIONS

1. Complete renal ischemia was produced in ninety-five kidneys of eighty-four cats by clamping the renal pedicle. Following the re-

establishment of the circulation of the ischemic kidney, a significant rise in blood pressure occurred in eighty-three experiments.

2. The same experiment was performed on four rats and five dogs. A significant rise occurred in all but one of these experiments.

REFERENCES

1. Taquini, A. C.: Liberacion de substancia hipertensora en el riñon completamente isquemiado, Rev. Soc. argent. de biol. **14**: 422, 1938.
2. Prinzmetal, M., Lewis, Harvey A., Taggart, J., Wilkins, H., and Drury, D. R.: The Effect of Transplanted Ischemic Kidneys and of Temporary, Complete, Renal Ischemia Upon the Blood Pressure of Rabbits, AM. HEART J. **20**: 525, 1940.
3. Prinzmetal, M., Lewis, Harvey A., and Leo, Sidney D.: The Etiology of Hypertension Due to Complete Renal Ischemia, Proc. Soc. Exper. Biol. & Med. **43**: 696, 1940.
4. Prinzmetal, M., Lewis, Harvey R., and Leo, Sidney D.: The Etiology of Hypertension Due to Complete Renal Ischemia, J. Exper. Med. **72**: 763, 1940.

SOME PRINCIPLES GOVERNING THE SUPPLY OF BLOOD TO THE MYOCARDIUM IN OCCLUSIVE ARTERIAL DISEASE

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INTRODUCTION

MANY anatomists since Lower[1] have called attention to the peculiar arrangement of the muscle fibers and bundles in the ventricles of mammalian hearts. At first these bundles were divided into superficial and deep layers, running in different directions. Gerdy,[2] Mall,[3] Flett,[4] Robb,[5] and others have attempted to define several muscle groups, which can be demonstrated consistently, and have given them the status of independent muscles. Four of these, the superficial bulbospiral, the superficial sinospiral, the deep bulbospiral, and the deep sinospiral muscles, are easily demonstrable anatomically, and sometimes another, the scroll muscle, can be separated.

Robb[6] has shown by experimental ligation of arterial branches of the coronary tree and by electrocardiographic studies that there is much evidence that these muscles function as correlated units.

The author showed, in 1939 (Lowe[7, 8]), that, under certain circumstances, naturally occurring pathologic processes, such as infarction and scar formation in the ventricular walls, follow the anatomic distribution of these muscle bundles. Recently, two additional instances in which the arrangement of pathologic processes depended on the anatomic distribution of the muscles in the ventricular wall have been recorded (Lowe[9]). In one case, petechiae resulting from acute leucemia occurred only in the superficial bulbospiral muscle; in the other, rupture of the left ventricle occurred, and the path of the escaping blood was in part determined by the muscle planes of the ventricular wall.

It may be assumed from this evidence, anatomic, physiologic, and pathologic, that the separate muscle bundles represent units of structure which constitute a physiologic, as well as an anatomic, entity.

In the investigations reported in this paper, colored gelatine masses were injected under carefully controlled pressures into the coronary circulation through both the main coronary arteries and their smaller branches. The results which were obtained show that it is possible by this means to inject the vessels in a single muscle.

These results also indicate that there is a general principle which governs the blood supply to cardiac muscles in the presence of occlusive arterial disease.

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METHODS

The hearts were obtained several hours after death. A large cannula was tied into the aorta (*Ao.*, Fig. 1), and the coronary circulation was perfused with normal saline solution through the aorta. The pressure applied was always of the same order as the arterial blood pressure had been during life. After the vessels had been thoroughly washed out with saline solution, a small cannula was inserted into a branch of one of the coronary arteries and directed centrally. The pressure of the perfusing fluid at the cannula in this small branch was recorded on an open manometer.

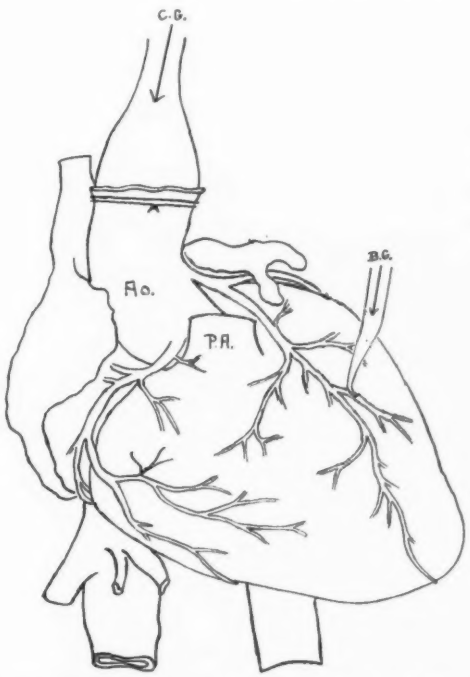

Fig. 1.—Drawing to illustrate injection experiments. Note cannulae in aorta (*Ao*) and coronary branch, clear gelatine (*C. G.*) in aortic cannula, and blue gelatine (*B. G.*) in branch cannula. (Modified from Cloquet's Anatomy.)

After the pressure in the aorta and in the coronary branch had been recorded, the small cannula was reversed so that it pointed in a peripheral direction, and the branch was tied off proximally. A clear gelatine mass* was then injected into the aortic cannula under various pressures. Simultaneously, a blue gelatine mass was injected into the branch cannula under a measured pressure which often corresponded to that recorded during the perfusion, but was capable of being raised or lowered at will (Fig. 1).

Throughout the whole experiment, all parts of the apparatus and the heart containing gelatine were kept immersed in a physiologic saline bath at a temperature of 37° C.

After the injection had been continued long enough to ensure that it was complete, the tubes to the cannulae were clamped while the gelatine was still under

*The blue gelatine was prepared in the manner described in a previous paper (Lowe[10]). The clear gelatine was made in the same way but no pigment was added; the viscosity was subsequently adjusted by the addition of distilled water and checked in a viscometer.

pressure, and the heart and cannulae were immersed in cold, 10 per cent formalin solution. After twenty-four hours' fixation, the heart was cut into transverse slices about 2 cm. thick and fixed for an additional twenty-four hours. The pieces were then kept in hydrogen peroxide solution until the muscle was completely bleached.

From these sections, drawings showing the extent of the injections were prepared in a manner similar to that previously described (Lowe[7]).

SPECIMENS

*Specimen No. 67.**—This was an adult heart into which a clear gelatine mass was injected through the aorta, and blue gelatine through a branch of the left circumflex coronary artery. The injection pressure was 180 mm. Hg in each case.

Examination of the sections showed a wedge-shaped area of injection extending from the pericardium to the endocardium in the posterior portion of the lateral wall of the left ventricle. The injected area also extended from the site of the injection (*A*, left middle section, Fig. 2) to the apex of the heart. There was no sign of laminar injection of the ventricular muscle, except in the section nearest the apex.

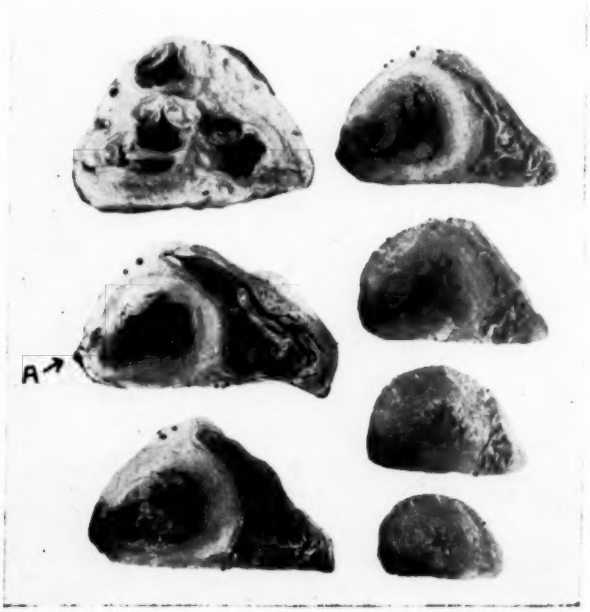

Fig. 2.—Photograph of the sections of an injected heart (Specimen 67), showing wedge-shaped, dark, injection areas in most sections.

Specimen No. 69.—This specimen was obtained from an adult with generalized atheromatous changes in the arteries. The pressures pertaining to the coronary arteries and aorta were ascertained by perfusion before the injection of the gelatine masses. A pressure of 190 mm. Hg in the aorta (clear gelatine) was combined with a pressure of 50 mm. Hg in the first branch of the anterior descending coronary artery, into which a blue gelatine mass was injected. After fixation, the heart was cut into the usual transverse slices.

When the sections were examined, a laminar scar was seen in the wall of the left ventricle, extending from near the base of the ventricles to near the apex

*The specimen numbers refer to hearts which were examined in this and other investigations.

(Fig. 3*a*). Its greatest lateral extent was at a point about halfway between the base and apex, where it extended backward from the anterior edge of the lateral wall for approximately one quarter of the circumference. Above and below this region it gradually became narrower. At the point of its greatest thickness, the scar involved 80 per cent of the thickness of the wall of the left ventricle and was separated from the endocardial surface by a layer of muscle 1 or 2 mm. thick. Above this point the scar was dense, but, below it, it became less well defined and was interlaced with muscle bands.

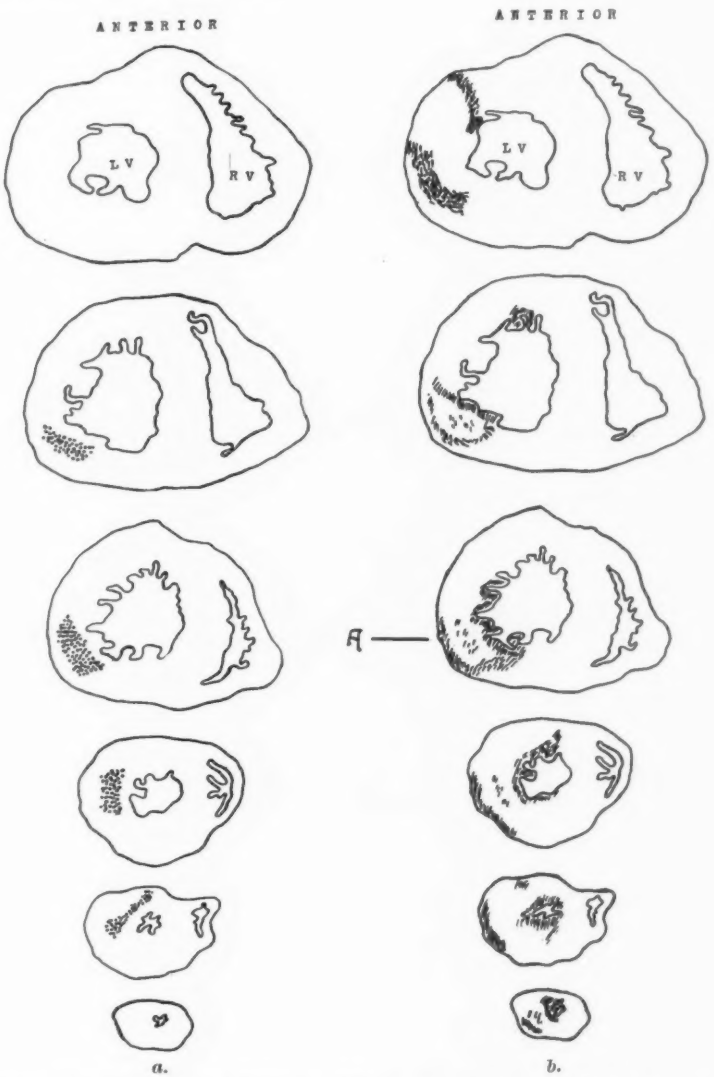

Fig. 3.—Drawings showing extent of scar (dotted areas, 3*a*) and injected areas (shaded areas, 3*b*) in Specimen 69. (Both diagrams on the same outlines, and may be superimposed.)

The blue gelatine mass extended up and down the ventricular muscle in laminae, and, in addition, some of it penetrated the papillary muscles and the scar tissue.

Some blue mass was also seen in the coronary artery above the point of injection (*A*, Fig. 3*b*); apparently, it had passed through collateral branches.

One of the laminae of blue mass was on the pericardial aspect of the ventricular muscle; it was a few millimeters thick and occupied a lateral position, as illustrated in Fig. 3*b*. Running from base to apex, it united at the apex with the other lamina, which was subendocardial and in the papillary muscles. This lamina stopped short of the base by about one-eighth of the distance from apex to base. In addition to these two laminae, there was some blue dye in the scar tissue. At the level of the injection of the blue mass (*A*, Fig. 3*b*) a wedge-shaped area of this mass extended through the whole thickness of the ventricular wall.

Specimen No. 70.—This preparation was obtained by injecting gelatine masses into a heart from a 14-year-old child. One cannula was inserted into the aorta and another into the first branch of the anterior descending coronary artery, a short distance from the junction with its parent vessel.

The pressures recorded during the preliminary perfusion were 120 mm. Hg in the aorta and 80 mm. Hg in this particular branch.

The injection was started with a pressure of 60 mm. Hg on the blue mass and no pressure on the clear gelatine in the aorta, and the blue mass ran easily into the coronary vessels distal to the cannula. However, when the pressure on the clear mass in the aorta was raised to 120 mm. Hg, the blue mass was forced back up the cannula until none could be seen in the coronary vessels. It was necessary to raise the pressure on the blue mass to 80 mm. Hg in order to obtain any flow into the coronary vessels against the pressure of the clear gelatine coming through the collateral circulation. By raising and lowering the pressures appropriately, it was possible to move blue gelatine up and down the vessels at will.

Finally, an injection was made with a pressure of 120 mm. Hg on the clear, and 80 mm. Hg on the blue, mass. The heart was then fixed, sectioned, and examined in the usual manner.

Near the site of injection, the blue mass had filled the capillaries in a wedge-shaped fashion from pericardium to endocardium (*A*, Fig. 4). As the sections were followed towards the apex, the injected area formed two laminae. One lamina was subpericardial; the other, subendocardial. At the apex these two laminae joined (lowest drawing, Fig. 4). Above the site of injection (*A*, Fig. 4) there was some injection of the ventricular muscle which was at first limited to the wedge shape, but nearer the base it assumed a laminal form. The whole of the injected region was on the lateral and posterior aspect of the left ventricle.

Specimen No. 71.—This heart was obtained from an adult who had had a systolic blood pressure of 220 mm. Hg and had died from cerebral hemorrhage.

Blue gelatine was injected into the first branch of the anterior descending coronary artery in accordance with the technique described. The pressures employed were the same as those applied previously during perfusion, i.e., 220 mm. Hg on the clear gelatine in the aorta, and 150 mm. Hg on the blue mass in the first branch of the anterior descending coronary artery.

After fixation, the heart was cut and examined. The injection in the region of the cannula extended throughout the whole wall in a wedge shape (*A*, Fig. 5). It extended in a superficial lamina from this point toward the apex, where it fused with a subendocardial lamina. There was also some injection of the papillary muscles of the left ventricle, extending to the base of the heart, i.e., to a level higher than the site of the injection (Fig. 5).

Specimen No. 65.—Blue gelatine was injected into the anterior descending coronary artery at about its mid-point. The pressure on the gelatine masses in both the aorta and the branch was 180 mm. Hg. The result was a wedge-shaped area

of injection extending through the thickness of the anterior ventricular wall, but showing some indication of a laminar pattern in the intraventricular septum.

Specimen No. 66.—Blue gelatine was injected into the first large branch of the anterior descending coronary artery, and clear gelatine into the aorta. The pressure on both masses was 180 mm. Hg. The result was a wedge-shaped area of injection in the lateral wall of the left ventricle, extending from the site of injection to the apex and through the whole ventricular wall. There was no sign of a laminar pattern.

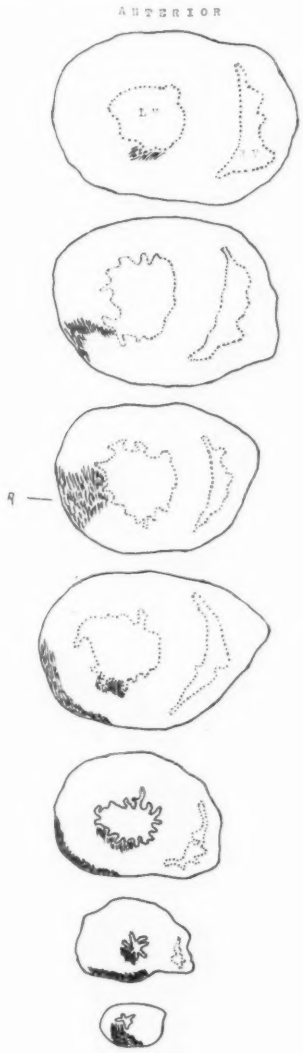

Fig. 4.

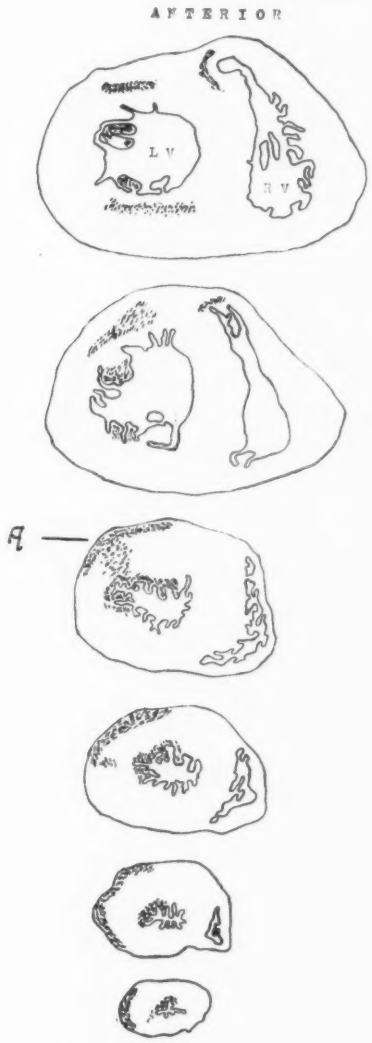

Fig. 5.

Fig. 4.—Drawing showing extent of injection in Specimen 70.
Fig. 5.—Drawing showing extent of injection in Specimen 71.

In all cases it was noticed that the extent of the blue injection masses increased after the hearts had been in hydrogen peroxide for some time.

Also, in the drawings the injected areas have been shown as if they had clear-cut boundaries. At their lateral extremities, however, it was usual to find a series of islands of injected tissue extending further than is indicated, but in the same general plane.

COMMENTS ON THE EXPERIMENTS

There are several observations to be made from these experiments. First, if the blue mass is injected into a branch of the coronary system under the same pressure as that previously ascertained by perfusion, the injected area of heart muscle is laminar and follows closely the anatomic relationships of the muscle concerned. The injection is not, however, completely confined to the one muscle, for at the level of injection a wedge-shaped area of muscle is supplied with gelatine. This closely parallels the distribution of petechiae in the superficial bulbospiral muscle in a case previously reported (Lowe[9]). In that instance there was a wedge-shaped area of hemorrhage near the base of the heart, as well as muscle distribution of hemorrhages.

The second observation is that, if the pressure on the gelatine in the branch is greatly in excess of that normally pertaining to that branch, the injected material spreads outside the limits of any one muscle and fills a wedge-shaped area which involves the whole thickness of the wall over a considerable longitudinal extent. The gelatine passes through anastomotic channels into a block of tissue without regard to its fine anatomic muscle arrangement.

Third, in the experiment on Specimen 70, it was shown that, by varying the pressure on the gelatine in the branch, the extent of the area injected could be altered quickly within wide limits. This means that in this case there was a free anastomosis between the portion of the coronary circulation supplied by the branch and the rest of the system, and that the normal balance depends simply on pressure relationships between the various branches of the coronary arteries. The fields normally supplied by various vessels represent a dynamic, not a static, equilibrium between those vessels. With a gross discrepancy of pressures (Specimens 65 and 66), the blue gelatine in the branch spreads over a very large field, whereas, with balanced pressures (Specimens 69, 70, and 71), the injection is confined to specific muscles.

It also appears that metabolic activities in the muscle had not ceased at the time of the injections, for the assumption that these changes were continuing is the only simple explanation for the increase in extent of the blue areas when the muscle slices were immersed in hydrogen peroxide. Apparently, the Berlin Blue dye acts as a hydrogen acceptor and is decolorized in the process. When the tissue is killed by fixation and the colorless dyestuff is exposed to an oxidizing agent, it regains the color. The bleaching process was not very powerful, for in the areas in which there were large quantities of blue dye there was no noticeable

reduction. It was only at the periphery, where the quantities of dye were small, that the change was seen.

The results obtained by injecting Specimen 69 show that in this heart the balances of the circulation were upset by the arterial changes which preceded the scar formation. This was revealed, first, by the marked decline of pressure between the aorta and the coronary branch (190 mm. to 50 mm.), which offers a sharp contrast with the normal decline of 120 mm. to 80 mm. in Specimen 70 and 220 mm. to 150 mm. in Specimen 71, in which arterial changes were not marked, and, second, by the fact that not only the muscle, but also the scar tissue was injected; the scar was in the deep bulbospiral muscle, which is not normally supplied by this artery.

Reviewing the injection experiments, we find that two distinct types of results are obtainable. First, if the injection pressure in the small artery is too high, a large area of tissue which is not confined to any one bundle, but extends through the whole thickness of the ventricular wall, is injected.

Second, if the pressures are correct, the injected area is laminar, with outlying islands of injected tissue which are mainly confined to one muscle bundle.

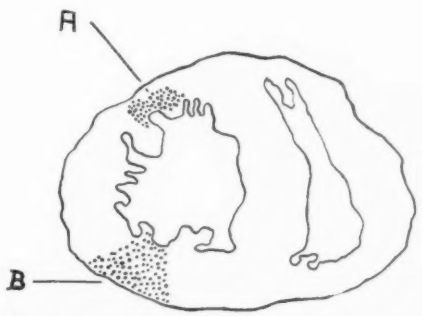

Fig. 6.—Drawing illustrating (*A*) massive scar, associated with thinning of the ventricular wall, and (*B*) massive infarct involving the whole thickness of the ventricular wall.

These injected areas bear a striking resemblance to the scars which are found in the ventricular muscle in arterial disease. These scars may be grouped into three distinct types, (1) that in which there has been large-scale destruction of the ventricular muscle, with subsequent replacement by fibrous tissue and frequently thinning of the ventricular wall (Fig. 6*A*); (2) that in which there is laminar scarring in the ventricular wall, sometimes associated with thinning of the wall (Fig. 7*A*); and (3) that in which the scar tissue is disseminated in islands throughout the ventricular wall (Fig. 7*B*). In addition to the scars, the large, wedge-shaped areas of infarction (Fig. 6*B*) which are found in some

cases of rupture of the ventricle must be included in the list of results of vascular obstruction.

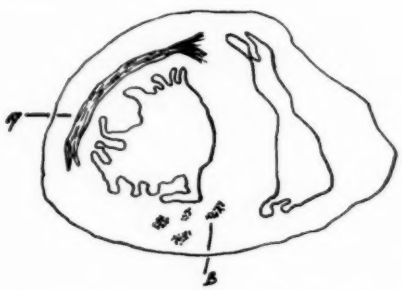

Fig. 7.—Drawing illustrating (*A*) a laminar scar in the ventricular wall and (*B*) disseminated islands of scar tissue.

The laminar injection areas, i.e., those limited to particular portions of individual muscles, correspond to the laminar scars described in detail by the author in previous papers.[7, 8] The wedge-shaped areas of injection resemble both the wedge-shaped scars (Fig. 6*A*) which are sometimes seen in the myocardium and the large areas of infarction (Fig. 6*B*) in ruptured ventricles and in cases of recent infarction which caused death. The outlying areas of injection are similar to the disseminated fibrosis that is so common in hearts which are the seat of gross atheromatous arterial changes.

DISCUSSION

Porter[11] wrote: "Everyone agrees that the coronary arteries anastomose. It is not the absence, but the character of the anastomosis, that is the basis of the present pathological teaching. The incontestable fact is that the anastomosis is too slight to permit a collateral circulation sufficient to keep a vascular area alive after the closure of the artery which supplies it."

The results of these experiments support Porter's statement that the vessels anastomose, but show that to say that a satisfactory collateral circulation cannot develop is correct only under very limited circumstances. Often a very efficient collateral circulation is formed. How much blood flows through an anastomotic vessel will naturally be determined by the pressure differences at the two ends of the channel. Normally, these differences are negligible, and, therefore, little blood will flow and the connections are overlooked (Fig. 8*a'*, *a''*, *a'''*). If, however, changes occur in the pressure distribution in the system, blood will flow through anastomotic channels, and they then become very obvious (Fig. 9*a''*, *a'''*). If blood flows through the connecting vessels, those into which it flows become dependent upon a new parent vessel for their supply of blood. Thus, when we consider the fields of supply

of various vessels, it becomes apparent that the supply of a given area of tissue may change.

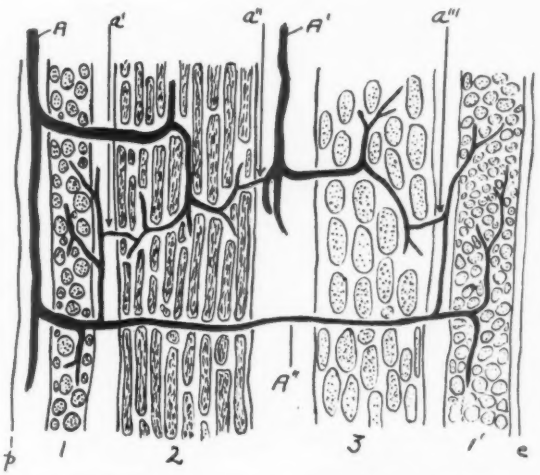

Fig. 8.—Diagram illustrating the normal arrangement of arteries among the muscle bundles of the ventricle.

p, pericardium; *e*, endocardium;
A, a subpericardial branch of the coronary arteries;
A', an intermuscular branch of the coronary arteries (see Fig. 10);
A'', an arterial branch running through the thickness of the wall (see Fig. 11);
a', *a''*, and *a'''*, nonfunctioning anastomotic channels;
1, the subpericardial layer of a superficial muscle;
1', the subendocardial layer of the same superficial muscle;
2 and *3*, layers representing two deep muscle bundles.

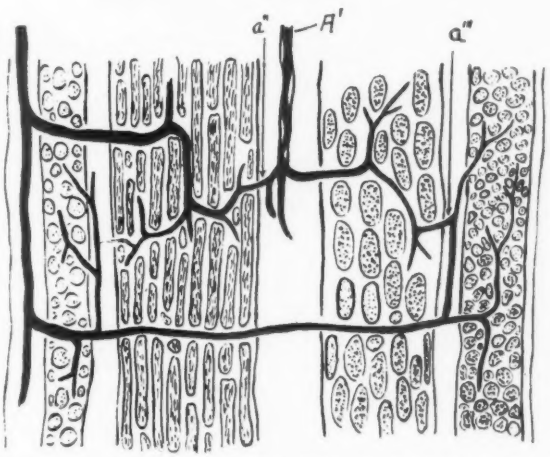

Fig. 9.—Diagram illustrating the redistribution of blood flow when one main vessel (*A'*) is partially occluded.

a'' and *a'''*, anastomotic channels now functioning.

We must therefore envisage the blood supply fields as the result of a dynamic equilibrium (Fig. 12); and the boundaries between the fields will move, perhaps considerably, if the normal pressure distribution is

altered (Fig. 13). Naturally, if pressure disturbances are sudden and great, it will take time for complete readjustment, partly because of spasm of the vessels distal to the block, and also because the small anastomotic arterioles must develop into arteries. If sufficient time is available, compensation by redistribution of the blood supply may be perfect.

The evidence in favor of this conception is very strong. The areas supplied by the right and left coronary arteries in the normal (child's) heart, as defined by injections, have been shown to vary according to the pressure balance on the two gelatine masses.[10] If only one main vessel is injected, the mass spreads considerably into the field supplied by the other vessel, whereas, if both are injected together, the two fields are quite clear-cut. In Specimen 70 a similar result was obtained with a small branch of the coronary artery; it was shown that the area of injection could be controlled almost at will by varying the pressures on the injection masses.

In the experiments reported by the author in 1937, hearts were injected after tying a branch of one coronary artery, in the hope that one muscle bundle would remain uninjected. The results were very inconclusive; annular uninjected areas of varying extent were sometimes observed, but there were no separate uninjected muscle bundles. The reason for this is that a dynamic equilibrium exists between the vascular fields. Tying off one branch merely diverts the gelatine mass into other channels which more or less completely inject the field of the occluded vessel.

The effect of occlusive arterial disease is to alter the pressure distribution between the branches of the coronary system, and, consequently, to change the route of the blood through the tissue as a whole. With gradual occlusion, complete or partial, of a vessel, there is ample time for the establishment of new routes (Figs. 9 and 13), but sudden occlusion partially deprives an area of tissue of its normal blood supply until the new routes open up to full capacity (Fig. 14). It may happen that, in this interval, death of tissue takes place, sometimes with disastrous results to the function of the organ as a whole. There are many observations which support this view that the vascular fields are subject to alterations. Gross[12] observed arterial anastomoses in old hearts, in which they were functioning as new routes of supply; Schlesinger[13] demonstrated them clearly by injecting hearts which were the seat of occlusive arterial disease; and the author (1937) showed that they existed, but did not function, in the child. It is well known that one main coronary artery may be found to be occluded at post-mortem examination, and yet the heart muscle may show no evidence of gross damage. There have also been several instances in which the heart continued to function for long periods after all three main coronary branches were occluded. Specimen 69 showed definite evidence of this

readjustment of vascular fields, for the blue gelatine entered the scar tissue in the deep bulbospiral muscle from a vessel which normally does not supply that muscle. In this instance the readjustment of the circulation was not made quickly enough to save the whole of the muscle from death, but the injection showed that sufficient blood flow from new routes was available to keep alive the scar tissue and the few remaining muscle fibers in that region.

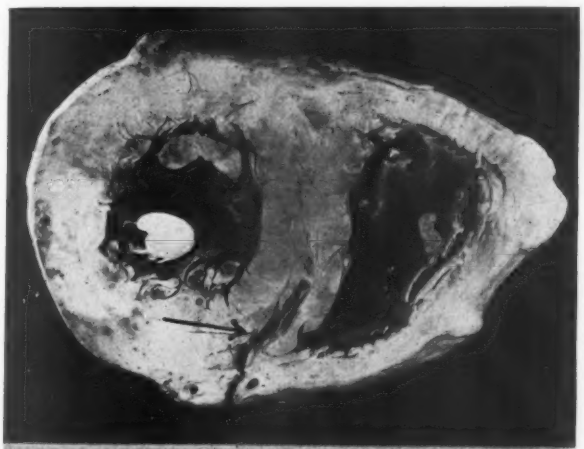

Fig. 10.

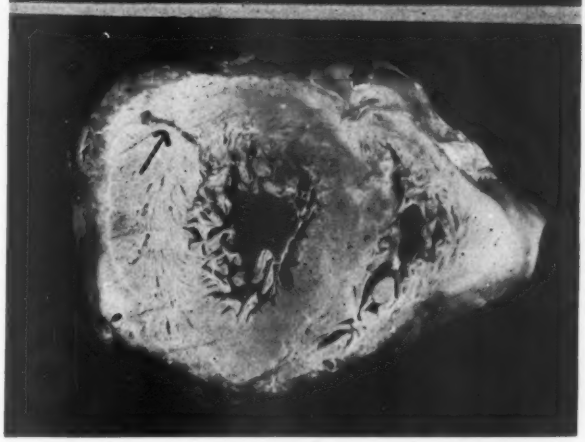

Fig. 11.

Fig. 10.—Photograph of an injected heart, showing arterial branch running between muscle planes.

Fig. 11.—Photograph of an injected specimen, showing arterial branch penetrating from the surface to the subendocardial layers.

Gradual occlusion of one or more vessels, therefore, merely brings about a redistribution of the blood supply. The effect of sudden occlusion is to deprive an area of tissue temporarily of the bulk of its supply. The extent of the area so deprived will of necessity be grossly different in a heart in which there have been gradual arterial occlusive changes and in one whose circulation is normal (Figs. 14 and 15). In the

former, the size of the area deprived of blood supply will depend on whether the occluded vessel took over a greater field in the redistribution, or was largely deprived of its field by the occlusion. If the occluded vessel had become a main channel to a large area, a large area

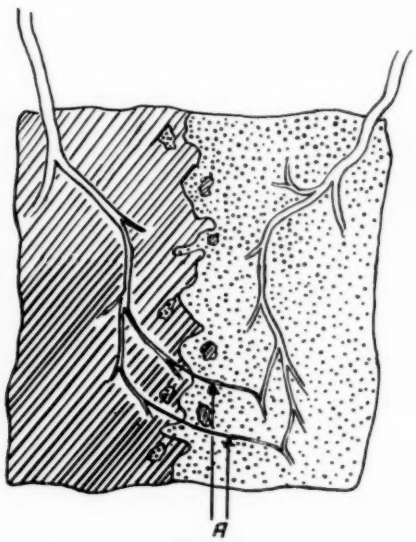

Fig. 12.

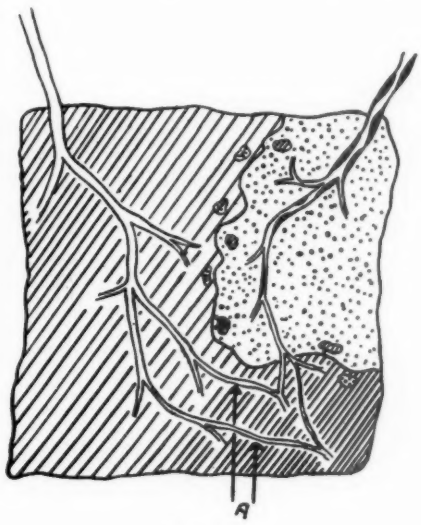

Fig. 13.

Fig. 12.—Drawing illustrating the normal fields of supply of two arteries. Nonfunctioning anastomotic vessels (*A*) shown. Drawing also illustrates the irregular and overlapping boundaries of these fields.

Fig. 13.—Drawing illustrating the change in fields of supply when one vessel is much more obstructed than the other. Anastomotic vessels now functioning (*A*).

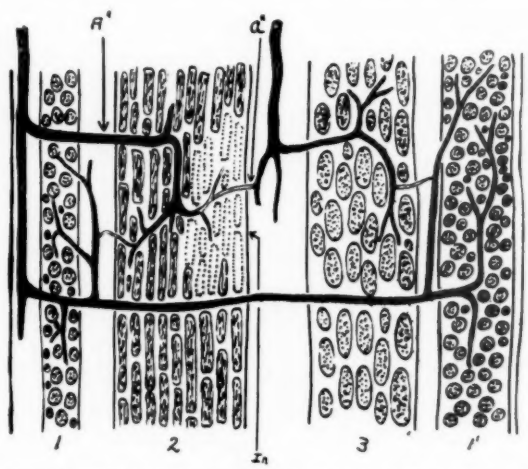

Fig. 14.—Diagram illustrating the effect of sudden obstruction of a small branch of the coronary artery *A''*. Because of spasm, the anastomotic channel *a''* is not functioning. The resultant infarct (*In*) is shown in dotted outline, and is confined to one muscle bundle (*2*).

will be rendered ischemic, whereas, if it were carrying very little blood, the area affected will be small. Sudden obstruction of a certain vessel

in a normal heart, e.g., by embolism, may produce an area of infarction (Fig. 14), but in a heart with occlusive arterial changes, occlusion of this vessel may produce almost no effect, or it may involve a vastly more extensive area than in the normal heart (Fig. 15). These areas vary from small, discrete islands to large, wedge-shaped infarcts which may involve the entire thickness of the ventricular wall, depending on which vessel is affected.

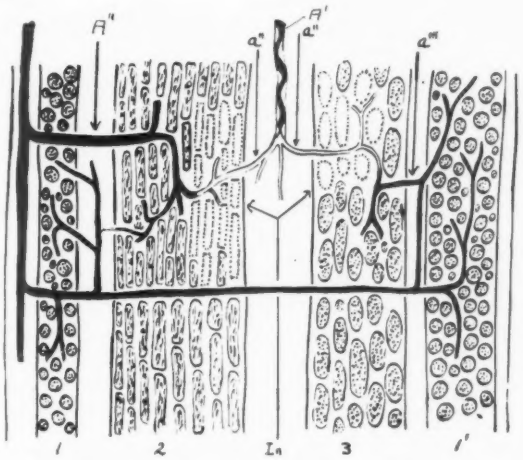

Fig. 15.—Diagram illustrating the effect of sudden obstruction of the same vessel as shown in Fig. 14, but in a system which is the seat of occlusive changes. The resultant infarct now involves two muscle bundles (*2, 3*). *A'*, artery almost completely occluded by obstructive changes; *a''*, anastomotic vessels which were carrying blood from artery *A''*, but are now deprived of blood supply by obstruction of *A''* branch. *a'''*, anastomotic vessel carrying some blood, but an insufficient amount to prevent the formation of an infarct (*In*).

Because there are distinct muscle layers and arteries both in planes between the muscle layers (Fig. 10) and passing directly through the muscle wall to the subendocardial layers (Fig. 11), the area of infarction may be confined to one muscle layer. It is also clear that, although obstruction of a vessel when the circulation is normal may produce an infarct limited to one muscle (Fig. 14), if there has been a readjustment of the circulation as a result of occlusion of arteries, obstruction of that same vessel may produce an infarct involving more than one muscle (Fig. 15). A similarity between laminar scars and laminar injections and between wedge-shaped injections and scars is therefore to be expected; the result will depend on the unpredictable vascular rearrangement.

There is, however, another possible and likely explanation for the multiple, discrete islands of fibrosis which are commonly seen in atheromatous hearts. If the occlusive changes are general throughout the larger vessels, there will be a reduction in the total quantity of blood available for distribution. In such circumstances, no rearrangement of paths will prevent the peripheral parts from receiving an inadequate

supply. These parts will therefore die and be replaced by islands of scar tissue (Fig. 16). The fact that there are islands of injected tissue at the periphery of the injected fields and that the dye is reduced by the metabolic activity in that region is evidence in support of this contention. It also suggests that, before death takes place, there will be a period of increased utilization of the oxygen available in the blood.

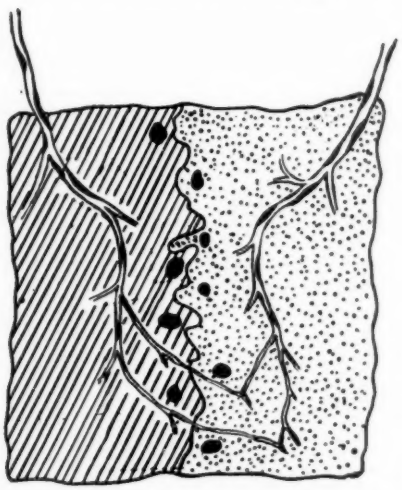

Fig. 16.—Drawing to illustrate the islands of dead tissue (black) at the periphery of the vascular fields when there is gradual, severe, and equal occlusion of both arteries.

The principle that there is a dynamic balance of blood flow between the branches of the coronary arteries therefore affords a simple explanation of the various types of scarring which occur in the myocardium. It also accounts for the discrepancies in the results obtained by various observers who have injected the coronary arteries with different substances.

APPLICATIONS

In as much as this conception of a dynamically balanced circulation is fundamental to the understanding of the pathologic changes in hearts which are the seat of occlusive arterial disease, it is not surprising that it also gives a clue to the meaning of the symptoms of this disease. Further, since it is a general principle, it should be of value in interpreting the results of arterial occlusion in any organ.

Considering first the heart, we find that clinical evidence indicates that there are varying types of symptom complexes. If the arterial changes result in gradual death of islands of muscle tissue, there will be a reduction in the amount of muscle available. The maximum effort of which the heart is capable is therefore reduced, and attempts to exceed this lowered maximum will be limited either by pain or dyspnea. An increasing extent of these lesions will lead to increasing severity of the

symptoms. Sudden vascular disturbances, such as thrombosis, will, if they cause infarction, produce a sudden lowering of cardiac reserve. The infarcts will vary in size from small areas to massive blocks of muscle, and the symptoms range all the way from those of a sudden, slight reduction of maximum output to severe attacks of pain and sudden death.

The manifestations of the occlusive arterial diseases of the leg are similar, namely, angina cruris, fatigability of the legs, and infarction of portions of the posterior tibial muscles as a result of vascular occlusion.

Consideration of the vascular lesions of the brain shows the same sequence. Pathologically, there are areas of degeneration and sclerosis which vary considerably in extent, and the large areas of infarction caused by cerebral thrombosis are well known. The corresponding symptoms vary from loss of memory, with the minor, diffuse lesions, to the syndromes caused by local lesions, such as paralysis agitans and the hemiplegia of sudden cerebral thrombosis.

In the kidneys, in cases of atherosclerosis, the lesions vary from small islands of scar tissue to massive infarcts.

Although this discussion has centered around the results of atherosclerotic vascular occlusion, the conclusions are equally applicable to syphilitic arteritis, thromboangiitis obliterans, or any other occlusive arterial process.

TYPE of INFARCT	RESULT
Large mass of tissue involving whole thickness of ventricular wall	Sudden death Rupture when infarct softens
Large mass of tissue not involving whole thickness of wall	Sudden death Rupture when infarct softens Aneurysm of ventricle
Portion of one muscle	Aneurysm of ventricle Linear scar (varying degree of myocardial inefficiency)
Disseminated islands of tissue	Disseminated islands of scar (varying degrees of myocardial inefficiency)

Fig. 17.—Table showing the consequences of various types of cardiac infarction.

This study of myocardial scars leads to some conclusions concerning the events which follow the sudden obstruction of a coronary artery (Fig. 17). The production of an infarct involving the whole thickness of the ventricular wall will lead, if death does not ensue immediately, to rupture of the ventricular wall when softening takes place in the infarct. An infarct which does not involve the entire thickness of the ventricular wall will produce a weak spot in the wall when it softens

and will lead to rupture if the overlying muscle layers are not strong enough to resist the intraventricular pressure. This was well illustrated in the case reported recently by the author.[9] If rupture does not occur at this weak point, there will be gradual stretching of the scar, leading to aneurysm formation, which carries with it the liability to subsequent rupture and endocardial thrombosis. The infarction of a large portion of one of the deep muscles, giving rise to the laminar scars which are often seen in cases of aneurysm of the ventricle, may be taken as a particular example of this type of lesion.

The degree of myocardial impairment depends not only on the absolute amount of muscle destroyed, but also on its anatomic situation. Robb, Hiss, and Robb[14] showed that the superficial muscles were dynamically relatively less important than the deep muscles.

Unless we have a way to ascertain the type and extent of the infarction, and the muscles concerned therein, the outcome of coronary occlusion is unpredictable. It seems probable that electrocardiographic studies may provide the means of identifying types of infarction. For many years classical abnormalities in the electrocardiogram have been recognized as indicating anterior or posterior wall infarction (Bohning and Katz[15]). More recently, Robb and Robb[16] have attempted to identify the type of tracing associated with individual muscle lesions. Blood pressure measurements may be helpful in estimating the outcome, either by indicating that a deep muscle bundle is involved or that the pressure throughout the whole coronary system is lowered, which would mean that the blood supply to areas not primarily involved in the infarction was deficient.

If all of this information were available, it would be possible to say, with respect to any particular coronary occlusion, that aneurysm formation, rupture of the ventricle, or impairment of cardiac function was, or was not, likely. Until such information is obtainable, we cannot give an accurate prognosis. Even if it were we could never say that there would not be another occlusion at a subsequent date.

In recent years there have been many attempts to supply a new collateral circulation to the heart which is the seat of occlusive arterial disease, either by producing adhesions between the pericardial layers, or by attaching extracardiac tissues (skeletal muscle) to the ventricular wall. The object in both cases is to develop anastomoses between coronary and extracardiac arteries. Whether or not this objective will be achieved depends on the pressure relations in the vessels concerned, and King[17] has shown that the pressure distribution is not always favorable to the development of such a collateral circulation.

CONCLUSIONS

The evidence that there is a series of separate muscle bundles in the ventricular wall of the mammalian heart, and that they function in-

dependently, is now very strong. The proof no longer rests entirely upon anatomic dissections and preparations, but is supported by injection studies of the coronary circulation and by investigation of the distribution of pathologic processes in the ventricular walls.

Not only do the results of the injection experiments confirm the anatomic studies, but they also indicate that a balanced anastomotic coronary circulation is a normal phenomenon.

It is further obvious that this principle of a balanced anastomotic circulation is applicable to occlusive arterial disease in any organ.

That the efficiency of an anastomotic circulation which develops when an artery is occluded depends on the state of the collateral vessels is no new thesis. However, it is not generally recognized that there is a continuous dynamic balance between the flow in these various vessels. This conception of a dynamic balance in the circulation explains many phenomena which occur in the heart in occlusive arterial disease.

It is also clear that, at post-mortem examination of hearts which are the seat of obstructive arterial disease, it is necessary to examine all of the vessels, not merely those which normally supply the region where there is infarction or fibrosis, as the case may be.

Study of cardiac scarring also indicates clearly that the prognosis in cases of myocardial infarction varies considerably. The type of infarction which is likely to occur is entirely unpredictable because the character of the redistribution of the blood supply resulting from the arterial occlusion is unknown.

SUMMARY

1. A series of experiments in which colored and uncolored gelatine masses were injected into the coronary circulation is described.

2. In these experiments it was demonstrated that a gelatine mass could be injected into the vessels of a single ventricular muscle.

3. It was also shown that, in order to achieve laminar injection, a careful study of the pressures in the coronary system was necessary.

4. Several measurements of pressures at various points in the coronary circulation were recorded.

5. It is deduced that there is a dynamic balance between the fields of supply of branches of the coronary arteries.

6. This principle is used to explain the method of formation and the significance of the various types of myocardial scars in arterial occlusive disease.

7. The conclusions are applied in a general way to the pathologic study of occlusive arterial disease in all organs and are correlated with the clinical observations.

8. Some of the prognostic problems in cases of coronary occlusion are discussed.

I wish to acknowledge my indebtedness to Professor MacCallum for the help and encouragement which he has given me in this investigation. My thanks are also due the members of the technical staff, who rendered much assistance.

REFERENCES

1. Lower, R.: Tractus de Corde, 1669 (Quoted in Quain's Anatomy, 1929, vol. 4, pt. 3, p. 73).
2. Gerdy: Recherches, Discussions, et Propositions, Paris, 1823 (Quoted by Cloquet, J.: Anatomie de l'Homme, Paris, 1828, vol. 3, p. 500).
3. Mall, F. P.: Am. J. Anat. **11**: 211, 1910-11.
4. Flett, R. L.: J. Anat. **62**: 439, 1927.
5. Robb, Jane S.: M. & Prof. Women's J., March, 1934.
6. Idem: Proc. Soc. Exper. Biol. & Med. **31**: 761, 1934.
7. Lowe, T. E.: J. Path. & Bact. **49**: 195, 1939.
8. Idem: M. J. Australia **2**: 491, 1939.
9. Idem: M. J. Australia **1**: 826, 1940.
10. Idem: Roy. Melbourne Hosp. Clin. Rep. **8**: 79, 1937.
11. Porter, W. T.: J. Exper. Med. **1**: 55, 1896.
12. Gross, L.: Blood Supply to the Heart, Oxford M. Pub., 1921.
13. Schlesinger, M. J.: An Injection Plus Dissection Study of Coronary Artery Occlusions and Anastomoses, AM. HEART J. **15**: 528, 1938.
14. Robb, J. S., Hiss, J. G. F., and Robb, R. C.: Localization of Cardiac Infarcts According to Component Ventricular Muscles, AM. HEART J. **10**: 287, 1935.
15. Bohning, A., and Katz, L. N.: Arch. Int. Med. **61**: 241, 519, 1938.
16. Robb, J. S., and Robb, R. C.: Am. J. M. Sc. **197**: 7, 18, 1939.
17. King, E. S. J.: Discussion on Coronary Ischaemia, M. J. Australia **2**: 524, 1939.

A NEW PIEZOELECTRIC MANOMETER TO RECORD INTRACARDIAC PRESSURES AND FOR THE SIMULTANEOUS RECORDING OF INTRACARDIAC ELECTROGRAMS*

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I.

FOR the study of the effect of various agencies on the heart, it is frequently convenient to have simultaneous records of the electrical and mechanical manifestations of its activity. This can be accomplished in a number of ways; for instance, the classical form of the Frank-Wiggers manometer may be made to register pressure on part of the same film on which the electrocardiogram is being recorded. Although this method is capable of yielding accurate records, it is laborious and necessitates opening the chest of the animal and using artificial respiration. When two string galvanometers are at hand, a more convenient procedure is available. The pressure variations may be transformed into electrical changes that can be recorded with one of the instruments. In recent years the piezoelectric effect of Rochelle salt crystals has been utilized extensively for recording sounds and other vibrations, and it seemed probable that this method might also be employed to record pressure changes.

The apparatus (Fig. 1) which we devised for accomplishing this consists of a rugged housing (Fig. 1*B 1*), inside which a large slab of crystal (Fig. 1*A c*, and *B 3*) is secured at one end. The free end of the crystal is in contact with a diaphragm (Fig. 1*A b*, and *B 5*) which closes the end of a long, narrow cannula (Fig. 1*A a* and *B 7*), filled with saline solution. If certain limits are not exceeded, such a crystal can be made to produce charges which are accurately proportional to the pressure distorting it.

The problem of constructing an appropriate recording system involves two other considerations. First, the crystal must be connected to the string galvanometer in such a way that the static charges which develop under pressure are not dissipated too rapidly. This is accomplished by using a vacuum tube with a low grid current to couple the crystal to the galvanometer. Since the voltages developed by the crystal are relatively high, no amplification of the effect is needed, and a very simple circuit may be used.

The coupling of the crystal to the source of pressure is equally important. In this case the problem is to construct a system with a sufficiently high-frequency response. For this purpose a long cannula filled with salt solution is used. The end in contact with the crystal is closed with a very tightly drawn rubber diaphragm; the end which is inserted into the heart is left open. To prevent clotting, heparin is added to the salt solution with which the cannula is filled. The cannula is of small enough bore and sufficient length to be inserted into the heart by way of the jugular vein or the carotid artery. The cannula with its diaphragm is constructed

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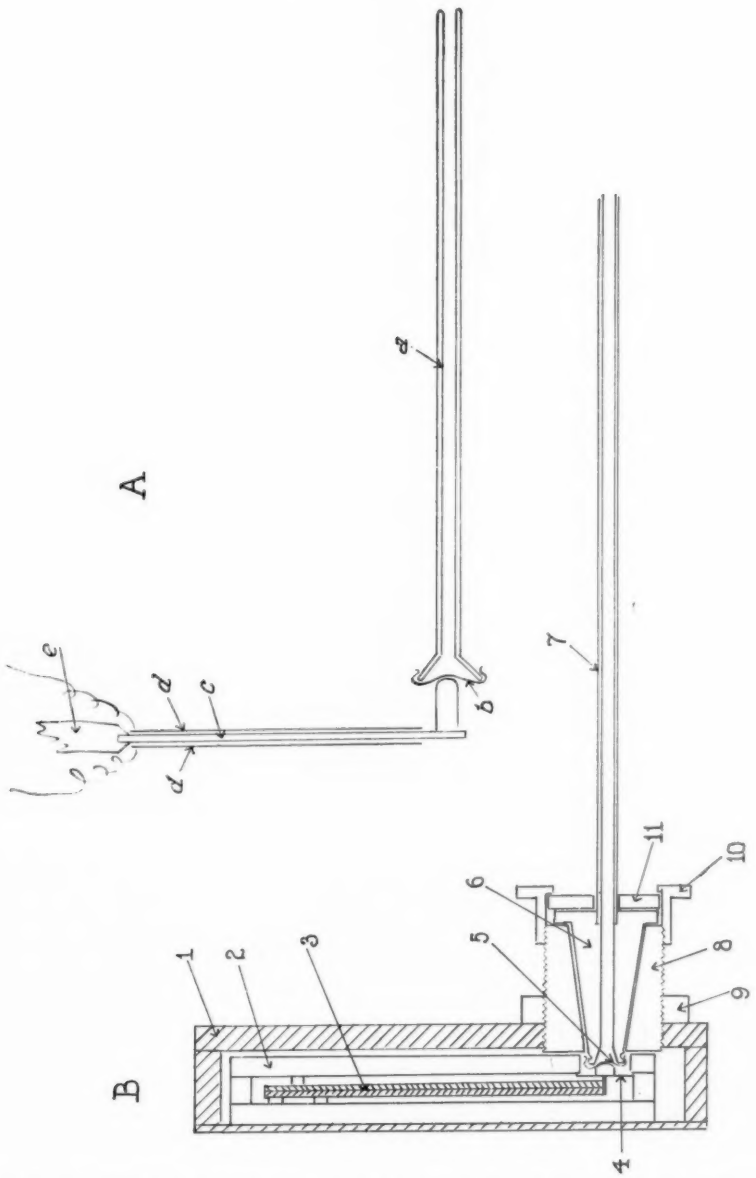

Fig. 1.—Piezoelectric manometer. *A*, Schematic drawing to illustrate the principle of operation. *a*, Cannula filled with salt solution; *b*, Distensible membrane which covers the end of the cannula and impinges upon a button connected to the free end of the crystal, *c*; *c*, slab of Rochelle salt crystal, held rigidly at one end by support *e*, and covered on both surfaces by plates of tin foil, *d*. When the open end of *a* is inserted into a chamber of the heart, the pressure changes which occur within it cause a distention of the membrane, *b*, which, in turn, bends the slab of crystal, *c*. This deformation of the crystal causes its faces, which are covered by the tin foil, to assume opposite electric charges. If certain limits are not exceeded, these changes are proportional to the amount of deformation of the crystal, and, consequently, to the changes in pressure. *B*, Plan showing the actual construction of the manometer. *1*, Rigid duraluminum outer case; *2*, fiber inner case; *3*, crystal; *4*, diaphragm closing fiber case to protect crystal from moisture; *5*, very tightly drawn rubber diaphragm, closing end of cannula; *6*, the brass cone which forms the end of the cannula makes possible a very rigid connection between the cannula and the case of the crystal; *7*,

as a single unit (Fig. 1*B* 5, 6, and 7) which is readily detachable from the crystal housing. It is filled with salt solution by means of a capillary pipette, and freed of air bubbles. Since the bore is so small that there is no danger of spilling the fluid, it is as easily manipulated as an empty cannula. The crystal housing, which is relatively large, need not be attached until the cannula is in the heart. A number of cannulae of different sizes are prepared at the beginning of the experiment, so that the appropriate size may be selected after the vessels are exposed.

Hamilton, Brewer, and Brotman[1] have discussed at length the frequencies attainable through narrow cannulae, and they probably adequately demonstrated their contention, but their work has not escaped criticism. It will not be redundant, therefore, to discuss from a different point of view the factors upon which the frequency responses of cannulae depend.

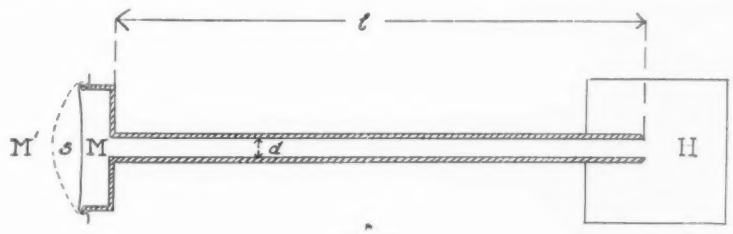

Fig. 2.

The cannula of length l (Fig. 2) is closed at one end by a distensible membrane (M), and connected to a chamber (H) in which the pressure varies. If the pressure in H is increased from P_1 to P_2, the membrane will assume the form M', and, consequently, an amount of fluid designated by s must pass through the tube. The speed with which this transfer can take place is the limiting factor in the frequency response of the cannula.

The principal forces which tend to retard the flow of fluid through the tube are inertia and friction. When small cannulae are used, the inertia of the fluid can be neglected, because it decreases with the bore. According to King's formula, the retarding effect of friction is $h_f = K\frac{l}{d^{1.25}}V^{n*}$. K is a constant which depends upon the roughness of the tube; l and d are the length and diameter of the tube, and V is the mean velocity in centimeters per second. If this formula is expressed in terms of s, the amount of fluid passing through it in time t (the time taken for the pressure to change from P_1 to P_2), it becomes $h_f = 4K\frac{ls^2}{td^{5.25}}$.

From the last equation it is apparent that an increase in length of the cannula (l) increases the resisting force, but that a decrease in diameter (d) increases it much more because this factor appears as the fifth power. Both of these effects can be counteracted, however, by a sufficiently great decrease in s (the distensibility of the membrane), which appears as the second power. Although it is clear from the equation what factors are involved, the precise extent to which a decrease in diameter can be compensated for by a decrease in the distensibility of the diaphragm cannot

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the cannula is made of silver and insulated with baked-on lacquer, save for the tip; 8, the receptacle for the conical end of the cannula is threaded, so that the contact between the diaphragm closing the end of the cannula and the button connected to the free end of the crystal can be adjusted; 9, a lock nut to hold the receptacle rigidly once the correct position is found; 10, a threaded collar with an opening large enough so that it can be slipped over the conical end of the cannula after it has been inserted into the animal's heart; 11, A slotted disk which can be slipped around the cannula after it is in place to engage the threaded collar.

*n is usually 2, but may vary from 1.75 to 2.08.

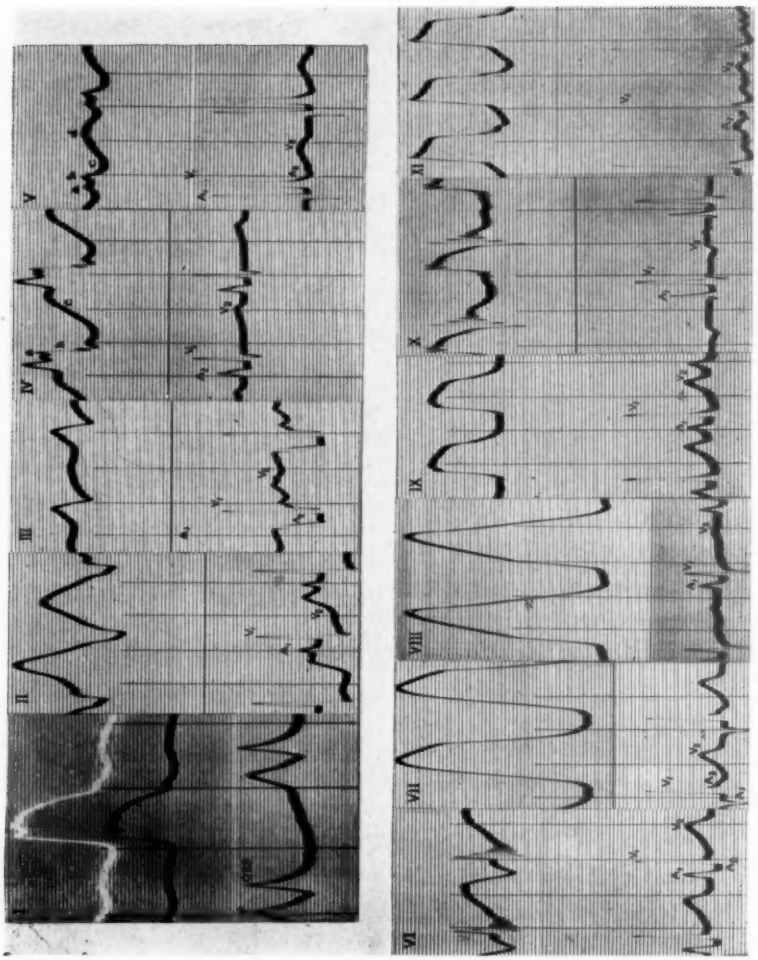

Fig. 3.—*I*, A comparison of curves obtained with piezoelectric and Frank Wiggers manometers. Top (white) curve, femoral pulse of a dog as recorded by the Frank-Wiggers manometer; middle (black) curve, femoral pulse recorded by the piezo-electric manometer. The bottom curve is a simultaneously recorded electrocardiogram. *II*, Curves which exhibit the effect of inserting the cannula too far into the right ventricle of a cat, so that its tip came in contact with the wall. The pressure curve is deformed (cf. *VII*, *VIII*, and *IX*), and the ventricular electrogram tends to be of a monophasic type (the take-off of the T wave is below the isoelectric line). *III*, Curves which exhibit the effect of inserting the cannula too far into the right auricle of a cat, so that its tip came in contact with the auricular wall. The pressure curve is deformed (cf. *IV* and *V*), and the auricular electrogram tends to be of the monophasic type (A_2). *IV*, Typical records from the right auricle of a cat obtained with an uninsulated cannula. The pressure curve (upper) shows an initial spike, *a*, caused by contraction of the auricle, followed, after a brief interval, by a sharp fall, *b*, and a subsequent, gradual rise, *c*, which are caused by ventricular activity. The electrogram (lower curve) resembles an electrocardiogram. Auricular activity is registered by an upward spike (A_1), followed by the two components of the ventricular complex, namely, V_1 (= QRS) and V_2 (= T). *V*, Records from the right auricle of a cat obtained with a cannula which was insulated, save for the tip. The pressure curve resembles *IV*, except that there is an additional positive phase, *d*, caused by auricular filling. In the electrogram (lower curve) the auricular response (A_1) is diphasic and of much greater voltage than *IV*. It resembles the intrinsic responses which are obtained when an electrode is placed in direct contact with the exterior of the auricle. The regression process of the auricle (A_2) is superimposed upon V_1, which is the initial ventricular response. *VI*, When curves are recorded with an uninsulated cannula in close contact with the tricuspid valve of a cat, the first heart sound appears in the pressure curve. *VII*, Curves recorded with an insulated cannula in the right ventricle of a cat; A_1, the auricular accession process, is downward, as is the intrinsic deflection from the

be predicted from it, but must be ascertained experimentally. The experiments of Hamilton, et al.,[1] have established the fact that cannulae much smaller than it has been necessary to use to enter the hearts of dogs and cats by way of the vessels in the neck can be employed without distorting the curves when the distensible diaphragm is a thin silver plate. The displacement (s) of such a diaphragm is very small. Since even less movement of the diaphragm is required to operate the crystal than Hamilton requires for his optical method of recording, his experiments are applicable to the cannulae which are used with the piezoelectric recorder.

A convenient record of electrical activity to pair with the pressure curve is one which emphasizes the effect of the chamber that is being explored. This is particularly desirable in the case of the auricle, for, in ordinary electrocardiograms, the electrical effects of its activity are so small that no detailed analysis of them is possible. Since Wilson and his collaborators[2, 3] have shown that, when one electrode is very close to the heart, it is affected predominantly by the muscle closest to it, this result is easily accomplished by using the tip of the cannula as the exploring electrode. To this end, the cannula is made of silver and insulated, save for the tip, with baked-on enamel. The exposed tip is rendered nonpolarizable by giving it a coat of silver chloride. A silver plate, inserted beneath the skin of the animal's hind leg, serves as the indifferent electrode. An added advantage of using the tip of the cannula as the exploring electrode is that any impingement of the heart wall that might occlude or partially occlude its orifice is at once betrayed by the appearance of a monophasic response in the electrical record (Fig. 3*II* and *III*).

The complete assemblies, including cannulae and crystals, have inherent frequencies in excess of 100 per second, as indicated by the usual tests, but since the ability of this instrument to record the slow components of the curves must also be checked, a direct comparison was made with the classical form of the Frank-Wiggers manometer by inserting the cannula of the crystal recorder into the femoral artery of a dog, and that of a Frank-Wiggers manometer into a branch close enough to the main stem so that the tip protruded into it. In this way the mouths of the two cannulae were brought very close together. Practically identical curves were obtained from the two instruments (Fig. 3*I*). The cannula used on the crystal manometer in this instance was the same length, but smaller in diameter, than those used to enter the heart.

II.

As the cannula is passed down the jugular vein of an animal, the pressure curves gradually increase in size until a typical intra-auricular pressure curve is obtained (Fig. 3*IV* and *V*). These curves are similar in contour to those of previous observers.[4] They nearly always show some rapid vibrations at the time of the occurrence of the heart sounds, particularly the first. When the cannula is pushed into the immediate vicinity of the tricuspid valve, these vibrations become marked (Fig. 3*VI*). When the cannula passes through the valve into the ventricle, the character of the curve changes abruptly into that of a typical intra-

exterior of the heart at the auriculoventricular junction. A_2, The auricular regression process, is upward and continued with V_1, which is diphasic: the second component is large and downward. *VIII*, Curves obtained with an uninsulated cannula in the right ventricle of a cat. V_1 is diphasic. *IX*, Curves from the right ventricle of a cat. V_1 is representative of the more usual monophasic type. *X*, Curves obtained with an insulated cannula in the aorta of a dog, near the aortic valve. The vibrations of the second heart sound can be seen in the pressure curve. A_1 resembles intrinsic deflections from the region of the sinus node. *XI*, Curves obtained when an insulated cannula is passed into the left ventricle of a dog. V_1 is monophasic.

ventricular pressure curve which usually shows no trace of the vibrations of the first heart sound (Fig. 3*VII*, *VIII*, and *IX*).

When the cannula is passed down the carotid artery, the carotid pulse which is recorded usually shows some trace of the second heart sound. When the cannula is close to the aortic valve, these vibrations are of considerable amplitude (Fig. 3*X*). After the ventricle is entered, the character of the curve abruptly changes to the intraventricular type (Fig. 3*XI*).

In the electrogram obtained from the interior of the right auricle the auricular complex is large, and both parts of it can be made out, i.e., the rapid accession process (A_1, Fig. 3*V*) and the regression or T deflection (A_2, Fig. 3*V*). The latter is usually combined with the ventricular part of the electrogram, but in many instances it can be seen quite clearly. A_1 is usually diphasic, and the first phase is downward. When the cannula is near the junction of the great veins with the auricle, this downward phase is small (Fig. 3*V*); near the auriculoventricular junction it is larger; and in the upper part of the ventricle (Fig. 3*VII*) it (the downward phase) alone remains. The auricular regression deflection, as a rule, consists of a small, rounded, downward or upward deflection (A_2, Fig. 3*V* and *VII*) between V_1 (QRS) and V_2 (T). In some of the earlier experiments no insulating lacquer was used on the outside of the cannula; under these circumstances A_1 consisted of a single upward phase (Fig. 3*IV*) which did not change in shape when the cannula was pushed into the ventricle (Fig. 3*VIII*).

III.

Usually, electrograms from the interior of the two ventricles are similar; they consist of a single upward (negative) deflection (V_1) of large amplitude, followed by an upward, downward, or nearly isoelectric T wave (V_2) (Fig. 3*IX* and *XI*). Occasionally, from the right ventricle, curves are obtained in which the initial component is diphasic; the initial (V_1) upward deflection is followed by a downward (positive) deflection (Fig. 3*VII* and *VIII*). The ventricular complexes obtained when the cannula is still in the auricular cavity resemble those from the ventricle, except that they are not so large.

If the crystal in the manometer is deflected by subjecting the cannula suddenly to a change of pressure, the crystal faces become charged, but these charges will gradually leak away, even when the pressure is maintained constant. Since the time required for these charges to dissipate is ten seconds or more, the rapidly changing pressures which accompany cardiac contraction are recorded accurately, but constant pressures are not recorded at all. Consequently, only pulse pressures can be measured with this instrument. For many purposes, however, this is sufficient, particularly if information is desired about the force of cardiac contraction rather than changes in the peripheral resistance. Since the crystal, when deflected, behaves like a condenser discharging through a high resistance, the lower the voltage, the less the rate of discharge, so that,

if a relatively small voltage is produced by deflecting the crystal only slightly, the accuracy of the record is increased. Since only a few millivolts are required to give records of good amplitude, one may use very stiff diaphragms which not only improve the performance of the crystal, but increase the frequency response of the cannulae.

The pressure curves obtained from the three chambers of the heart which can be explored with this type of manometer are similar to curves obtained by other appropriate means.[4] The value of the new instrument depends on its convenience and on the fact that the animals can be left intact and their circulation not interfered with. It has the advantage over the method of Hamilton, et al.,[1] which may also be used without opening the animal's thorax if a needle is thrust through the chest wall into the heart, that there is less uncertainty as to the position of the cannula, and that the animal need not be near the recording camera. The animal can be studied in an adequate light without interfering with photographic recording.

Only one type of pressure curve requires special comment, namely, that obtained in the immediate vicinity of the heart valves. These records may show sound-like vibrations which are coincident with valve closure (Fig. 3*VI* and *X*). These vibrations may be artifacts caused by impingement of the valves on the end of the cannula, or may be produced by closure of the valves (the valvular component of the heart sounds). Against their being artifacts is the fact that plucking (setting into vibration) the end of the cannula produced only very small vibrations in the record. It was, of course, necessary to construct the manometer so that sidewise tugging on the cannula would not distort the records with artifacts caused by motion of the heart. Obviously, after the cannula has entered the ventricle, the valve leaflets must still be impinging upon the cannula, but, nevertheless, the vibrations disappear. Brewer, Hamilton, and Brotman[5] have shown that very abrupt changes in pressure may set the recording system into vibration if its inherent frequency is not sufficiently high, but pressure changes quite as abrupt as those which occur at these points in the curves are not always associated with sound-like oscillations, notably the sudden rise in pressure during the isometric contraction phase. The conception of Brewer, Hamilton, and Brotman that practically all cardiovascular sounds are in reality caused by sudden changes in pressure which set the recording system or the eardrum into vibration is probably too sweeping. There can be no doubt that the heart sounds, for instance, are really sounds (repeated oscillations) at the anterior chest wall, for microphones capable of responding to frequencies as high as 30,000 cycles per second always record them as sounds consisting of many oscillations. Sudden changes of pressure occur when the myocardium contracts or the valves close, and these changes, when they are applied to adjacent inert tissues, will inevitably cause them to vibrate. Therefore, the first and second

heart sounds cannot be considered as instrumental vibrations unless all of the tissue between the source of shock and the surface of the body is regarded as part of the recording instrument. Although the vibrations which are recorded from the immediate vicinity of the valves by means of this new manometer may be some sort of artifact, they may also reflect the vibrations, caused by closure of the valves, which are transmitted to the surrounding tissues as the valvular components of the heart sounds.

Electrograms from the interior of the heart have not been recorded frequently,[6] for, although they have considerable theoretical interest, they have been of little practical value because of the difficulty in obtaining them. Since they are easily obtained with this new device and may prove to be useful records of the heart's electrical activity, they should be discussed briefly.

Wilson, Macleod, and Barker[3] showed that electrograms taken with an exploring electrode in contact with the auricle show intrinsic deflections which are similar to those obtained from the ventricle, but they can be obtained only when the electrode is very close to the auricular surface, whereas intrinsic-like deflections can be obtained from the ventricle even through the chest wall.[7] The electrograms from inside the auricle which were recorded in the course of this study resemble those taken from comparable positions on the outside. This similarity is to be expected, because an impulse which spreads radially bears the same relative position to any electrode which it passes, whether the latter be inside or outside the auricle, and because the conductivity of the blood is not very different from that of the tissues which surround the heart.

These electrograms from within the auricle are larger than those which Brown[8] obtained with an electrode in the esophagus, but not quite as large as those obtained by the usual methods from the exterior surface. The auricular complexes of the curves obtained from the aorta near the aortic valve (Fig. 3*X*) resemble Brown's curves closely.

Although it is impossible to know accurately to what part of the auricular muscle the tip of the cannula is nearest, the change in the shape of the curves as the cannula is pushed through the auricle is what would be expected from the work of Wilson, Macleod, and Barker.[3] An early intrinsic deflection, with a small, preceding, downward deflection, is obtained when the cannula first enters the auricle by way of the superior vena cava and is near the sinus node (Fig. 3*V*, A_1). In the auriculoventricular region the intrinsic deflection is late, and, in the upper part of the ventricle, it disappears, and the complex consists of a downward deflection only (Fig. 3*VII*, A_1). Curves of an intermediary type (the upward and downward phases of which are more or less equal) are not usually obtained because the cannula passes through the auricle from the orifices of the great veins to the auriculoventricular region without coming in close contact with the mid-portion of the auricular wall.

Because it is too small, the P wave of the electrocardiogram has been of comparatively little use in studying the condition of the auricular myocardium. It has, for instance, been known for a long time that the P wave is only that part of the auricular activity which corresponds to the QRS group of the ventricular complex; the part corresponding to the T wave is usually too small to be seen.* In electrograms taken directly from the surface of the auricle, and in Brown's curves from suitable positions in the esophagus, the T portion of auricular activity can be readily identified. In normal curves from the interior of the auricle, this TA deflection (Hering), or auricular regression deflection (Macleod[11]), usually falls in the QRS complex, and, consequently, is not easily recognized (Fig. 3*V* and *VII*, A_2). It may, however, as Brown has pointed out, deform the S-T segment; it may also account for the failure of the curve to return to the isoelectric line during the P-R interval. As will be shown in a subsequent paper,[9] alterations in this process are often very easily seen, although, in its normal form, it is inconspicuous. That the auricular regression deflection is a delicate index of the effect of drugs and other agencies on the auricular myocardium has been shown by Cohn and Macleod,[10] and Macleod.[11] It is, therefore, particularly desirable to be able to study this deflection in mammals.

Wilson, Johnston, and Hill,[12] in their study of myocardial infarction, described electrograms which were taken from the interior of the ventricles. The QRS of their curves consists of a single, tall, upward (negative) deflection. This is the type of curve which is to be expected if the impulse is conducted rapidly over the endocardial surface by means of the Purkinje network, and subsequently spreads outward through the ventricular wall at a slower rate. The curves which were obtained from the left ventricle in the course of the present study were always of this form. Those from the right ventricle were usually of the same type, but occasionally, both in dogs and cats, curves were encountered which showed a prominent downward (positive) deflection after the upstroke. The explanation of this deflection is not at once apparent; its presence might seem to be inconsistent with the view of Wilson, Johnston, and Hill, but, by utilizing the same principles upon which their concept is based, and taking into consideration certain anatomic facts, one can account for it.

Wilson, Macleod, and Barker[13] have explained how the active process can be likened to an expanding shell, so polarized that its outward surface is positive and its inner surface is negative, spreading outward through the ventricular muscle. An electrode in the left ventricle (*L*, Fig. 4) is exposed only to the negative surface of this shell throughout the period of excitation. Although it is true that the septal portion of the right ventricular shell has its positive surface towards electrode *L*,

*Brown's paper[8] contains a full account of the history of this deflection.

the effect of this is always neutralized by the septal part of the left ventricular shell.

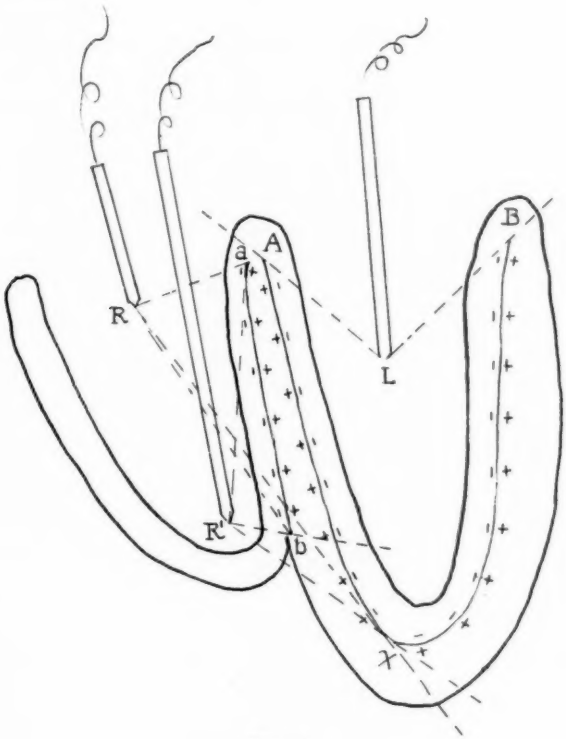

Fig. 4.

Electrode *R*, in the right ventricle, like *L* in the left, will be negative until its expanding shell reaches the outer surface of its lateral wall and disappears. Now only the septal portion remains. If the electrode is so placed (*R*) that this portion masks the positive surface of the left ventricular shell, it will remain negative or become zero, but, if the electrode is at R′, this septal portion of the right ventricular shell is insufficient to do this, and the electrode will become positive during the later part of the period of excitation.

SUMMARY

1. A manometer which simultaneously records intracardiac pressure curves and intracardiac electrograms is described.

2. Criteria are presented for estimating the accuracy of the pressure curves obtained with this apparatus.

3. Electrograms obtained with the noninsulated tip of the manometer as it is pushed progressively farther through the right auricle, the A-V valve, and into the ventricle demonstrate changes in the action currents corresponding to these levels.

4. The form of the electrogram obtained from within the ventricle is likewise discussed.

REFERENCES

1. Hamilton, W. F., Brewer, G., and Brotman, I.: Pressure Pulse Contours in the Intact Animal. I. Analytical Description of a New High-Frequency Hypodermic Manometer With Illustrative Curves of Simultaneous Arterial and Intracardiac Pressures, Am. J. Physiol. **107:** 427, 1934.
2. Wilson, F. N.: The Distribution of the Potential Differences Produced by the Heart Beat Within the Body and at Its Surface, AM. HEART J. **5:** 599, 1930.
3. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Distribution of the Action Currents Produced by Heart Muscle and Other Excitable Tissues Immersed in Extensive Conducting Media, J. Gen. Physiol. **16:** 423, 1933.
4. Wiggers, C. J.: Modern Aspects of the Circulation in Health and Disease, ed. 2. Philadelphia and New York, 1923, Lea & Febiger, p. 80.
5. Brewer, G., Hamilton, W. F., and Brotman, I.: Analysis of the Pulse Contour in Relation to Sound Production by Means of the Hydrogen Transmission Pulse-Recorder, Am. J. Physiol. **107:** 420, 1934.
6. Lewis, T., and Rothschild, M. H.: The Excitatory Process in the Dog's Heart. Part II. The Ventricles, Phil. Tr. Roy. Soc. London **206:** 181, 1915.
7. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Order of Ventricular Excitation in Human Bundle Branch Block, AM. HEART J. **7:** 305, 1932.
8. Brown, W. Hurst: A Study of the Esophageal Lead in Clinical Electrocardiography. Part I. The Application of the Esophageal Lead to the Human Subject With Observations on the Ta-wave, Extrasystoles and Bundle-Branch Block, AM. HEART J. **12:** 1, 1936.
9. Cohn, A. E., and Macleod, A. G.: The Effect of Acetylcholine on the Mammalian Heart, AM. HEART J. **21:** 356, 1941.
10. Cohn, A. E., and Macleod, A. G.: The Effect of Acetyl-Beta-Methylcholine on the Frog's Heart, AM. HEART J. **17:** 305, 1939.
11. Macleod, A. G.: The Effect of Certain Pure Digitalis-Like Glucosides on the Frog's Heart, AM. HEART J. **17:** 294, 1939.
12. Wilson, F. N., Johnston, F. D., and Hill, I. G. W.: The Interpretation of the Galvanometric Curves Obtained When One Electrode Is Distant From the Heart and the Other Near or in Contact With the Ventricular Surface. Part II. Observations on the Mammalian Heart, AM. HEART J. **10:** 176, 1934.
13. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Interpretation of the Initial Deflections of the Ventricular Complex of the Electrocardiogram, AM. HEART J. **6:** 637, 1931.

THE EFFECT OF ACETYLCHOLINE ON THE MAMMALIAN HEART

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I.

COHN and Macleod[1] have recently published a study of the effect of mecholyl and acetylcholine on the frog's heart. This study indicated the general mode of action of the drug, but, because choline derivatives are being used to a considerable extent clinically, particularly for the relief of attacks of paroxysmal tachycardia, and because the vagus mechanism of mammals is somewhat different from that of frogs, it seemed desirable to extend these studies to the mammalian heart. The effect of the drugs on the mechanical response of the heart could, furthermore, be more easily studied in the mammal because of its greater size.

The experiments were carried out on cats and dogs which were anesthetized with nembutal. The vagi were exposed and cut. Stimulating electrodes were attached to their peripheral ends. The cannula of the piezoelectric manometer, insulated except at its tip, which was described in a previous paper[2] was inserted into the heart by way of a jugular vein or carotid artery. The intracardiac pressure pulse was recorded, together with an electrogram derived from the tip of the cannula (exploring electrode) and a silver plate inserted under the skin of the left hind leg (indifferent electrode). Simultaneous records of the electrical and mechanical activity of the heart were obtained by this means without opening the animal's thorax. Records were obtained from the right auricle and the right ventricle by pushing the cannula through a jugular vein, and from the left ventricle by inserting it into a carotid artery. The drugs were administered intravenously. A dose of 1 to 2 mg. of acetylcholine (or about one-tenth that amount of mecholyl) was used for the average cat; dogs required 5 to 7 mg. of acetylcholine to obtain an equivalent effect.

II.

AURICLES

The earliest effect of stimulation of either vagus is slowing of the auricular rate. Accompanying, or immediately following, this, there is diminution in the force of auricular contraction (negative inotropic effect, Fig. 1 *II*, and *III*). At about the same time there are alterations in the auricular regression deflection (Fig. 1 *III*, A_2). After strong stimulation, marked shortening of this process occurs. The first observable effect, however, is a deformity of the segment which immediately follows the accession deflection (in the P-R region). It is difficult to be certain whether these changes precede, accompany, or follow the reduction in the force of contraction (the relationships differ in different experiments), but, in any case, they are nearly coincident with it. During the height of vagus action the amplitude of the whole of the auricular electrogram is reduced (Fig. 1 *III*). Prolonged stimulation occasionally initiates a brief paroxysm of auricular fibrillation.

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Acetylcholine produces similar results (Fig. 1 *IV*, *V* and *VI*), except that the changes are of greater magnitude; that part of the intra-auricular pressure curve which results from auricular contraction may entirely disappear (Fig. 2 *I*). The auricular regression deflection is

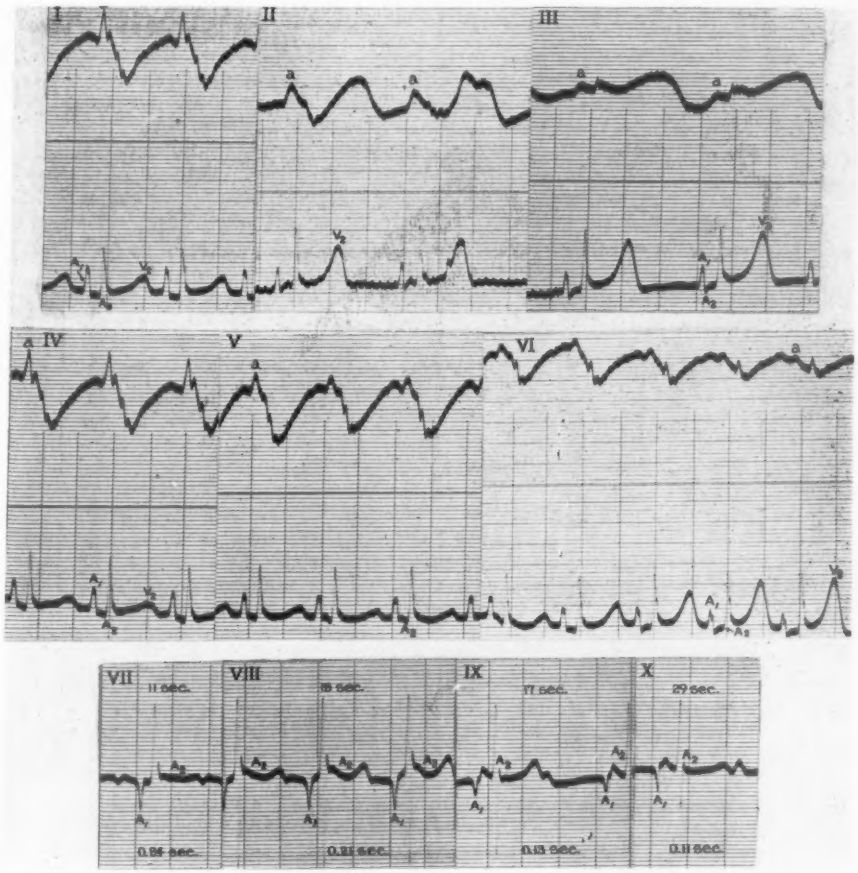

Fig. 1.—The effect of vagus stimulation and acetylcholine on the intra-auricular pressure curve during auricular contraction. *a* is the component of the auricular pressure curve caused by auricular contraction. A_1 represents the accession process in the auricular electrogram (P wave). A_2 is the regression process in the auricular electrogram (Ta wave). V_2 is the regression process of the ventricular electrogram (T wave). *I*, *II*, and *III* were taken before, during, and just after left vagus stimulation. *II* and *III* show a negative inotropic effect in the auricles. They also exhibit small ripples caused by escape of the stimulating current. *IV* was taken before injection of acetylcholine; *V*, seven seconds after the injection of 2 mg. acetylcholine; and *VI*, about ten seconds after injection. *VII*, *VIII*, *IX*, and *X* are electrograms obtained with the electrode inserted just inside the ventricle of a cat. They show shortening of electrical systole and a decrease in voltage of the auricular complex. The figures above each curve give the time after an injection of 2 mg. of acetylcholine; those below, the duration of electrical systole (from the beginning of the accession process to the end of the regression process).

greatly shortened (Fig. 1 *VII-X*). (Since heart block nearly always occurs at this stage, the changes in this process are undistorted by any ventricular effects.) The reduction in size of the entire auricular electrogram is marked (Fig. 2 *I*). Auricular extrasystoles usually occur

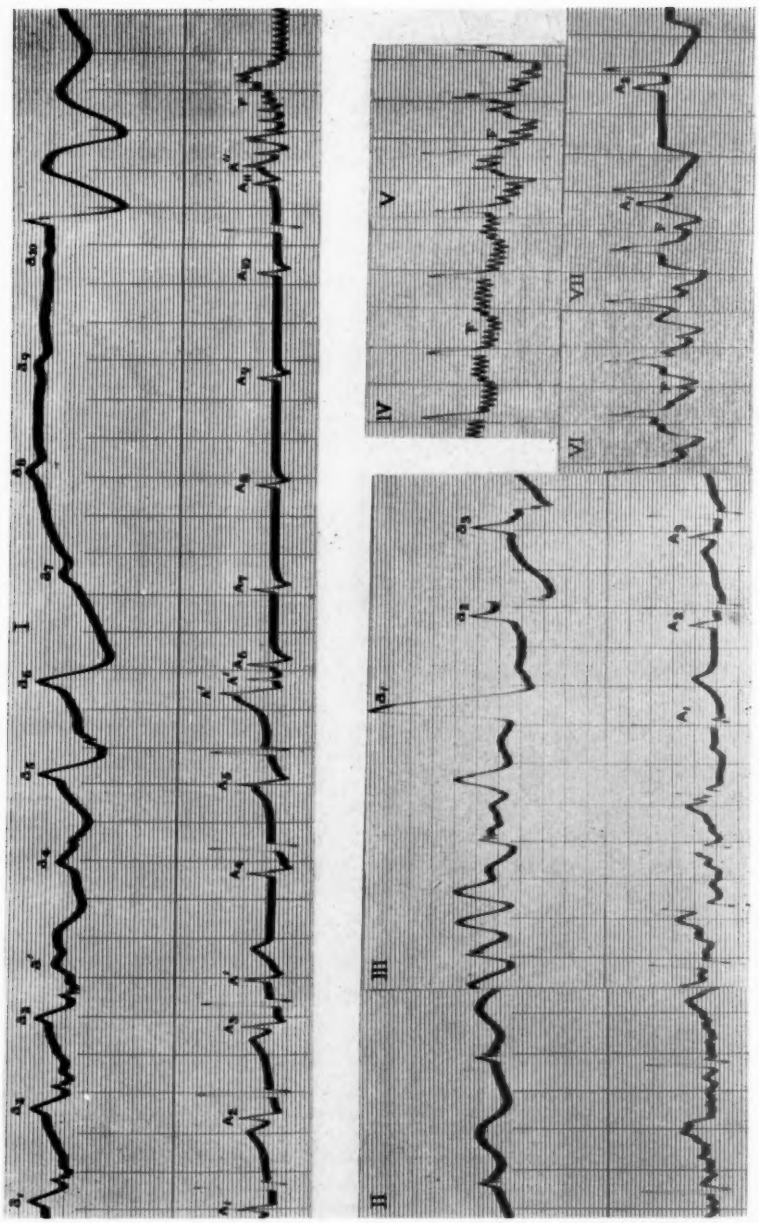

Fig. 2.—The occurrence of auricular fibrillation after the administration, intravenously, of acetylcholine to cats. a_1, a_2, etc., result from the rise in pressure caused by regular auricular contraction; and a^1, a rise in pressure caused by an auricular premature beat. A_1, A_2, etc., represent auricular electrograms, and *F*, waves of fibrillation. *I*, *II*, and *III* were taken from the same experiment. The tip of the cannula lay in the auricle, just beyond the entrance of the superior vena cava. *I* starts nine seconds after the injection of 2.5 mg. of acetylcholine, and shows a decrease in the vigor of auricular contraction (a_7, a_8, a_9, and a_{10}), great shortening of the auricular regression process (A_4-A_{10}), heart block following A_5, and auricular fibrillation following *A''*. *II* starts one minute after injection. *III* starts two minutes after injection. It shows the cessation of auricular fibrillation; the first normal auricular beat, A_1, is superimposed on a ventricular complex. It consequently produces a large rise in pressure, a_1. A_2 and A_3 are normal in contour, and give rise to normal pressure changes (a_2 and a_3) similar to A_1 and a_1 in *I*. *IV*, *V*, *VI*, and *VII*, from the same experiment, occur one, two, two and one-half, and three minutes after the injection of 3 mg. of acetylcholine. The abnormal rhythm is more in the nature of flutter (the waves remained regular); the decrease in rate can be easily followed, therefore, as the effect of the drug wears off. In *VII*, normal rhythm is resumed.

(Fig. 2 *I, A'*), and, if the dose has been large, one of these may initiate an attack of auricular fibrillation (Fig. 2 *I, A''*). This invariably starts as a series of rapid regular oscillations (state of rapid re-excitation) which sometimes are almost like those of alternating current (Fig. 2 *I* and *IV*, but, at other times, in each cycle, a more rapid and a slow component can be made out; these correspond to the accession and regression deflections. During the early stages of the attack ventricular contractions are infrequent, possibly because of the ineffectiveness of such small and rapid auricular stimuli, but more probably because of the coincident occurrence of auriculoventricular heart block. Then a stage usually occurs wherein the fibrillation waves are smaller and less regular (Fig. 2 *II*). As the effect of the drug wears off, the waves become coarser, until a state of impure flutter is reached; this finally gives way to normal rhythm (Fig. 2 *III-VII*).

Recovery from a single injection is apparently complete, but after repeated injections the heart weakens and the animal may die.

A-V STRUCTURES

As has been mentioned, when a sufficiently large dose is given, heart block, complete or partial, invariably occurs (Fig. 2*I*).

VENTRICLES

The effects of vagus stimulation on the ventricles are similar in kind to those produced on the auricles, but much less in degree. For the first few beats after vagus stimulation is begun, the intraventricular pressure curve may increase in size as a result of the increased diastolic filling (Fig. 3 *II*) which is made possible by bradycardia. Subsequently, the beats may diminish (Fig. 3 *III*) in strength, but this diminution is never so great as in the case of the auricles. The extent of the negative inotropic effect varies greatly in different preparations.

The ventricular electrogram also shows less dramatic changes than the auricular. Shortening of ventricular systole cannot be made out with certainty, but the terminal portion of the regression deflection (T wave) is definitely increased in height (Fig. 3 *III*). With prolonged stimulation there is possibly slight reduction in the voltage of the accession deflection (QRS). Ventricular fibrillation following vagus stimulation did not occur in any of the experiments.

Acetylcholine also exerts less effect upon these chambers than upon the auricles. An unmistakable reduction in the force of contraction usually occurs within a few cycles after the administration of the drug. This is accompanied by an increase in the height of the regression deflection (T wave, Fig. 3 *VI* and *VII*). There is often a definite reduction in the amplitude of the accession deflection (QRS). Ventricular extrasystoles occur occasionally, as does ventricular fibrillation. The latter usually persists long enough to cause death.

III.

A reduction in the intracardiac pressure pulse must indicate a negative inotropic effect unless the heart is inadequately supplied with blood or is contracting against reduced resistance. In the case of the auricles, after the administration of acetylcholine, it is inconceivable that they are inadequately supplied with blood, for both sinus bradycardia and heart block are present; the former allows adequate time for filling,

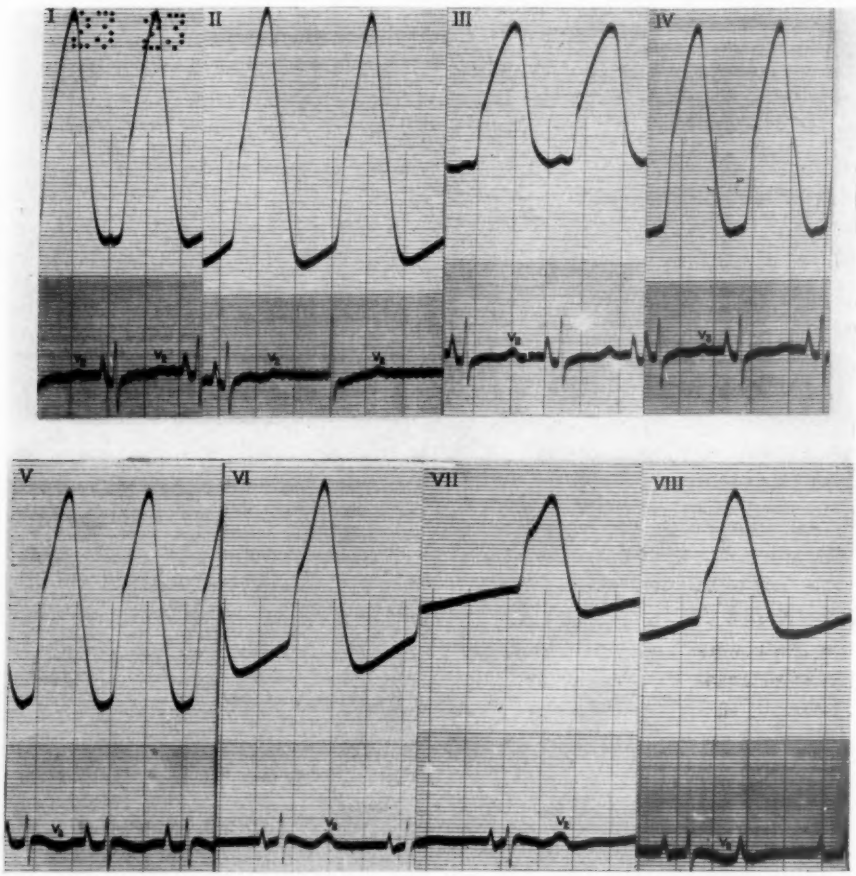

Fig. 3.—The effect of vagus stimulation and of mecholyl on the ventricles of cats. V_2 represents the regression deflection (T wave) of the ventricular electrogram. All of the curves are from the same experiment. *I* was taken before vagus stimulation. *II*, one second after the start of faradic stimulation of the peripheral end of the left vagus; it shows a transient increase in the amplitude of the ventricular pressure pulse (the stimulus artifact can be seen). *III*, fifteen seconds after the beginning of vagus stimulation and five seconds after its cessation; it shows a decrease in the ventricular pressure pulse and an increase in the height of the T waves. *IV*, fifty seconds after the cessation of stimulation; it shows the return of the mechanism to normal. *V*, *VI*, *VII*, and *VIII* occur before, five seconds, forty-five seconds, and one minute, respectively, after the injection of 0.15 mg. of mecholyl. The diminution in amplitude of the pressure pulse and the increase in height of the T wave are shown. In *VIII*, heart block is present. The electrogram has returned almost to its normal form, but the pressure pulse is still smaller than the control.

and the latter retards the removal of blood. These conditions also suggest that the auricles must contract against increased, rather than reduced, resistance. If the heart rate is slow enough so that the ventricles

have been passively filled with blood before the auricles contract, the resistance against which the auricles work is the venous pressure, and this is increased. It is, therefore, certain that the reduced amplitude of the auricular pressure curve indicates a genuine, negative, inotropic effect of the drug.

The reduction in the ventricular pressure curve can be less certainly ascribed to a depressant effect on the myocardium, for, although the slow ventricular rate assures adequate filling of the ventricles, a reduction in the diastolic blood pressure could reduce the resistance against which it contracts sufficiently to account for part or all of its reduction in vigor. Following stimulation of the peripheral end of the vagus, it is not likely that much lowering in blood pressure occurs, however. The change in the shape of the electrogram indicates, furthermore, that some myocardial change has occurred. It is probable, therefore, that both vagus stimulation and the administration of acetylcholine cause some reduction in the force of ventricular contraction. Drury[3] failed to observe a negative inotropic effect on the ventricles as a result of vagus stimulation with the Cushny myocardiograph, but the method of investigation used in this study is probably more sensitive and might show slight changes which the older method could not detect. In any case, the ventricular effects are of a different (much smaller) order of magnitude from those which occur in the auricles.

The alterations in the electrograms indicate, accordingly, an effect on the whole heart, but predominantly on the auricles. The shortening of electrical systole in the auricles is strikingly similar to that observed by Cohn and Macleod[1] in the frog's heart. Although estimations of the refractory period were not made in this series, it is well known from the work of Lewis, Drury, and Bulger[4] that vagus stimulation reduces the refractory period of the auricles. A considerable body of evidence indicates, furthermore, that the duration of electrical systole and of the refractory period vary pari passu. It may be concluded, therefore, that acetylcholine reduces the duration of the active state of the mammalian auricle, as well as that of the frog.

Such a quick return to the resting stage may indicate a simple acceleration of the recovery process. If this were the case, subsequent systoles should not differ in other respects from preceding systoles. But this is not the case, for the mechanical response of these beats is greatly reduced, as is the voltage of the accession (P) process. In other words, rapid recovery of excitability and conductivity occurs without proper recovery of the ability to contract. Except for a reduction in the length of electrical systole, a similar dissociation of the different functions of muscle occurs in the case of calcium deficiency (Mines[5]).

If the views of Macleod[6] are correct, an altered state of recovery would be expected to affect the voltages developed during activation. The reason for this inference is that, if the constitution of resting muscle

differs from normal, so, in all probability, does the potential difference between it and active muscle. Conversely, if this potential difference is less than normal, as it is after the administration of acetylcholine, it follows that resting muscle (under its influence) is less different from active muscle than is normal resting muscle; it has incompletely recovered. An increase in the duration of the excitation process (QRS interval) could also cause a reduction in the voltage which is developed, but no significant change in the duration of this process occurs.

No shortening of the electrical response in the ventricle is observed, but there is a definite increase in the height of the regression deflection (T wave). Such a change in the form of the T wave can be accounted for only by alteration in the course taken by the spread of complete recovery, i.e., a local alteration in the duration of electrical systole. It cannot now be demonstrated whether this particular local alteration results from an increase in duration in one locality or a decrease in another. The course of events in the auricle makes the latter seem the more likely, however.

Auricular fibrillation occurs frequently after the administration of acetylcholine. After electrical systole and, of course, the refractory period have been shortened, an extrasystole nearly always gives rise to a series of ectopic beats and frequently initiates an attack of fibrillation. This arrhythmia usually persists until the effect of the drug has worn off to a considerable extent. The occurrence of these paroxysms is probably to be accounted for by a reduction in the duration of the refractory period of the auricles, which facilitates the occurrence of circus rhythm.

Ventricular fibrillation occurred in two of the twenty-eight experiments. The doses of acetylcholine were not larger than those which are usually used. In both cases it persisted long enough to cause death. Since fibrillation of the ventricles is usually attributable to something which alters their refractory period, it is probable that the occurrence of this aberrant rhythm is additional evidence that the drug produces shortening of the refractory period in that part of the ventricles which it affects. The occurrence of this irregularity in cats and dogs under the circumstances of these experiments suggests that a large dose may induce it also in patients.

The most extensive clinical use of acetylcholine and its derivatives has been in the treatment of attacks of paroxysmal tachycardia. Since they have a strong effect on the auricles and only a weak one on the ventricles, it would be surprising if they were not much more efficacious in relieving paroxysmal tachycardia of auricular, than of ventricular, origin. This does, in fact, seem to be true in the few cases of ventricular paroxysmal tachycardia so treated which have been reported. Two patients with, presumably, ventricular paroxysmal tachycardia who were treated by Starr[7] were not relieved by the drug; nor, in a case

reported by Harvey[8] was acetylcholine efficacious. In the two cases reported by Stern,[9] mecholine was used, but without benefit.

It is probable that the beneficial effect of the drug in cases of paroxysmal tachycardia depends on its ability to shorten the refractory period, but quinidine, which is also useful in the treatment of this condition, has an opposite effect; it lengthens the refractory period. A similar paradox was noticed by Wilson and Wishart[10] in their study of the intravenous use of digitalis, which also shortens the refractory period, for the relief of paroxysmal tachycardia. They concluded that these drugs apparently terminate attacks in different ways unless they possess a common effect on a still unknown property of cardiac muscle. This alternative probably does not apply in the case of acetylcholine and quinidine, because, as Starr[11] has found, quinidine opposes the cardiac action of acetylcholine in animals, and, in patients, a previous dose of quinidine nullifies the effect of acetylcholine on paroxysmal tachycardia. The beneficial effects of the two drugs depend, therefore, on opposite properties.

To explain these contradictory effects it becomes necessary to conclude that the desired result is obtained, not by shortening, lengthening, or adjusting the refractory period to some optimal length, but as a mere consequence of changing it. If the refractory period changes, for example, more rapidly than the abnormal mechanism can adjust itself to the new circumstances, the aberrant rhythm will cease. In this connection it is noteworthy that the most effective drugs for this purpose act rapidly and are quickly eliminated. Digitalis is not an exception to this statement, for it seems to be effective only when a large dose is given intravenously. The prophylactic effect of quinidine may depend on the fact that a constant waxing and waning of the refractory period makes the establishment of an abnormal rhythm difficult. On this assumption, a choline derivative with a slower and more prolonged action would be less, rather than more, useful.

The effect of acetylcholine on the mammalian heart is, as this study shows, purely vagomimetic. Although its effect is more intense than that of stimulation of the peripheral end of the severed vagus, it is qualitatively similar. The failure of the drug to have as pronounced an effect on the ventricles as on the auricles is of particular interest.

If the effect of the drug were exercised directly on the myocardium, the ventricles should be as greatly affected as the auricles. If the drug acts not upon the muscle itself, but upon the vagus endings, this selective action is understandable, for it is well known that the ramifications of the vagi reach nearly all parts of the auricles, but only the upper part of the ventricles. This idea is supported by the fact that the frog's ventricle, which is well supplied with vagus endings, is as much affected by the drug as the auricle,[1] and by the observation of Cohn[12] that acetylcholine has no effect upon the chick embryo heart before vagus fibers have grown into it. Since Starr[11] has cast grave doubt on Dale's[13] concept that acetyl-

choline is effective after the vagus endings are paralyzed, it is probable that acetylcholine is not the effector substance elaborated by the vagus endings, but merely a substance capable of stimulating these endings.

CONCLUSIONS

By recording simultaneous intracardiac pressure pulses and electrograms, it has been shown that the effect of acetylcholine on the auricles is to reduce the force of contraction and the duration of the excited state, and frequently to produce fibrillation; it has a similar, but much slighter, effect on the ventricles. It is probable that it is more effective in auricular than in ventricular tachycardia and that it is of benefit in this condition because it changes the refractory period rapidly, rather than because it shortens it. It acts on the vagus endings, rather than on the muscle proper.

REFERENCES

1. Cohn, A. E., and Macleod, A. G.: The Effect of Acetyl-Beta-Methylcholine on the Frog's Heart, AM. HEART J. **17**: 305, 1939.
2. Macleod, A. G., and Cohn, A. E.: A New Piezoelectric Manometer to Record Intracardiac Pressures and for the Simultaneous Recording of Intracardiac Electrograms, AM. HEART J. **21**: 345, 1941.
3. Drury, A. N.: The Influence of Vagal Stimulation Upon the Force of Contraction, and the Refractory Period of Ventricular Muscle in the Dog's Heart, Heart **10**: 405, 1923.
4. Lewis, T., Drury, A. N., and Bulger, H. A.: Observations on Flutter and Fibrillation. Part VI. The Refractory Period and Rate of Propagation in the Auricle: Their Relation to Block in the Auricular Walls and to Flutter, etc., Heart **8**: 83, 1921.
5. Mines, G. R.: On Functional Analysis by the Action of Electrolytes, J. Physiol. **46**: 188, 1913.
6. Macleod, A. G.: The Electrogram of Cardiac Muscle. An Analysis Which Explains the Regression or T Deflection, AM. HEART J. **15**: 165, 1938.
7. Starr, I., Jr.: Acetyl-Beta-Methylcholin; Its Action on Paroxysmal Tachycardia and Peripheral Vascular Disease, With Discussion of Its Action in Other Conditions, Am. J. M. Sc. **186**: 330, 1933.
8. Harvey, A. M.: Origin of Paroxysmal Tachycardias as Determined by Oesophageal Electrogram, Ann. Int. Med. **11**: 57, 1937.
9. Stern, N. S.: Paroxysmal Ventricular Tachycardia; Report of 3 Cases; Mecholyl Used and Ineffective in 2, Ann. Int. Med. **11**: 519, 1937.
10. Wilson, F. C., and Wishart, S. W.: Effect of Intravenous Administration of Digitalis in Paroxysmal Tachycardia of Supraventricular Origin, AM. HEART J. **5**: 549, 1930.
11. Starr, I., Jr.: A Note on the Antagonism Between the Cardiac Action of Acetyl-Beta-Methylcholine and Acetylcholine and That of Quinidine, J. Pharmacol. & Exper. Therap. **56**: 77, 1936.
12. Cohn, A. E.: Unpublished investigations.
13. Dale, H. H.: Note on the Reversal of Vagus Action by Quinidine as Seen in the Heart of the Cat, Heart **9**: 87, 1921.

CONTRAST ROENTGEN VISUALIZATION OF COARCTATION OF THE AORTA

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THE roentgenologic diagnosis of coarctation of the aorta is based ordinarily upon indirect evidence, since the narrowed and the adjacent segments of the aorta often are difficult or impossible to demonstrate. Notching of the caudad edges of the ribs posteriorly is most characteristic but is demonstrable only occasionally. Changes in the cardiac silhouette due to left ventricular hypertrophy may occur. The size and pulsations of the aorta proximal to the coarctation vary and are of no definite value in differential diagnosis. In children particularly, these roentgen findings are difficult to demonstrate. The method of Robb and Steinberg[1] provides direct visualization of the great vessels and their branches as well as of the cardiac chambers. Hence it should be applicable to the elucidation of clinically obscure or atypical instances of coarctation. This possibility is referred to by Robb and Steinberg.[2]

In the following two cases coarctation of the aorta was suspected clinically. Demonstration of the actual coarctation and its precise localization was accomplished by contrast visualization. The fluorographic method described previously[3] was used in order better to identify the cardiac chambers and the aorta.

CASE REPORTS

Case 1.†—19570. E. M., a twelve-year-old boy, was seen in the Out-Patient Department of the Mount Sinai Hospital (service of Dr. Béla Schick) for the first time on March 16, 1940. He was and had been perfectly well except for childhood diseases. Seven months previously he was examined by a school physician who discovered cardiac murmurs and diagnosed rheumatic heart disease. For this reason the patient entered the clinic for examination.

Physical examination revealed the boy to be healthy in appearance though somewhat underdeveloped. The heart was enlarged to the left, the cardiac dullness reaching beyond the nipple line. There was a loud blowing systolic murmur over the lower right border which radiated to beneath the right scapula. A loud blowing diastolic murmur was heard over the second intercostal space and to the right of the sternum. This was transmitted to the neck.

Blood pressure determinations were as follows: Left arm 160/98; right arm 158/98; left leg 98/84; right leg 96/80.

Urine examination showed no albumin or sugar; the microscopic examination did not reveal any abnormalities.

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†The complete clinical data on this case are included in a forthcoming publication by S. Blumenthal and D. Davis, "Coarctation of the Aorta."

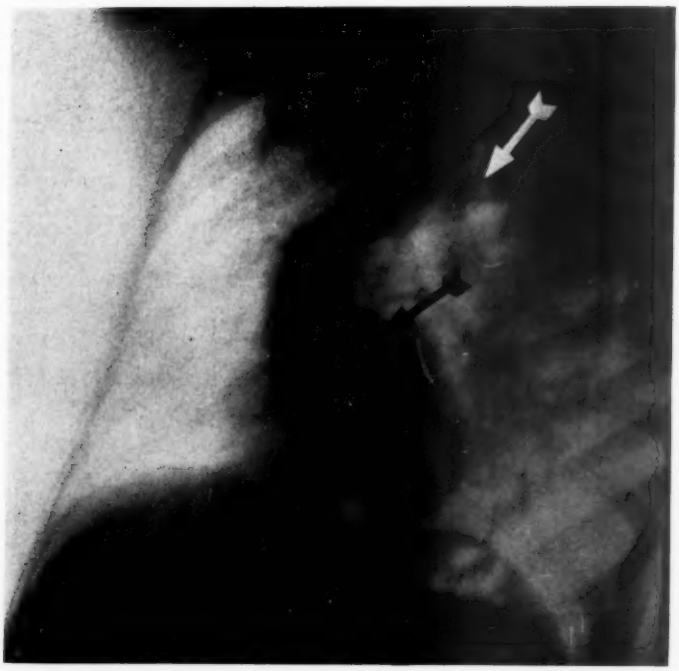

Fig. 1.—Case 1. White arrow points to coarctation. Black arrow points to aortic valve. There is considerable dilatation of the left ventricular cavity and the supracardiac portion of the aorta. There is marked dilatation of the arteries of the neck. The descending aorta is of normal caliber below the coarctation.

Fig. 2.—Case 1. The heart and large vessels are magnified because the patient moved away from the fluoroscopic screen during the exposures. However, arrow points to visualized right internal mammary artery.

Electrocardiographic examination showed no other abnormalities; there was no axis deviation.

A roentgenogram of the chest showed a somewhat enlarged heart which was globular in shape. The descending aorta was not visualized successfully. No abnormality was noted in the ribs.

Comment.—In this case, the clinical diagnosis of coarctation of the aorta was based upon the presence of a classical clinical picture. Corroborative roentgen evidence was lacking. Constrast visualization demonstrated the narrowed segment in the proximal descending aorta, a normal aorta immediately proximal and distal to the coarctation, moderate dilatation of the ascending aorta particularly in the supracardiac portion, and left ventricular dilatation and hypertrophy. There was a peculiar variation in the density of the ascending aorta, the significance of which was not determined. The dilated right internal mammary artery was also seen (Figs. 1 and 2).

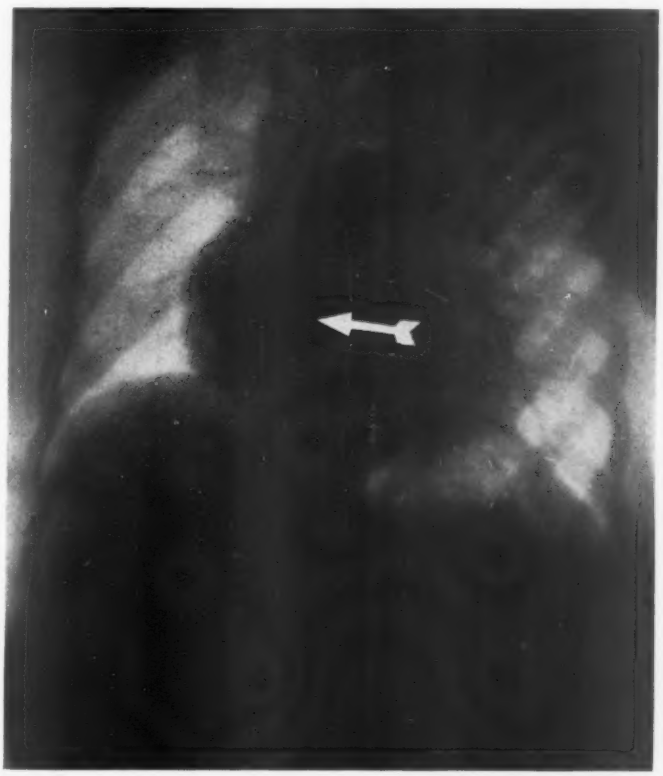

Fig. 3.—Case 2. Left oblique position. Visualization of the right auricle and right ventricle which are normal in appearance. The pulmonary artery is dilated. The interventricular septum reaches more to the right than usual because of enlargement of the left ventricle.

CASE 2.—L. C., 456241, a 23-year-old Jewish housewife, entered The Mount Sinai Hospital, service of Dr. B. S. Oppenheimer, on May 6, 1940, complaining of loss of weight, palpitation and neck enlargement during the previous two months. She had always been in good health but had been irritable, excitable and nervous. She had no headaches nor any cardiovascular symptoms. Two years previously her blood pressure, taken by a physician, was said to have been normal. Two months

previous to admission when she began to perspire easily, lose weight and experience palpitation, a physician found a markedly elevated blood pressure.

Physical examination showed moderate exophthalmus, lid lag and stare. The fundi revealed changes consistent with myopia. The thyroid was enlarged diffusely and a bruit was heard over it. The heart was enlarged to the left and a loud, high pitched systolic murmur was heard at the apex and over the pulmonary area which radiated to the back and lumbar region. P_2 was greater than A_2. The heart rate was 120-130 per minute, the rhythm regular. No intercostal pulsations were noted. There was a fine tremor of both hands. Repeated blood pressure determinations in both arms averaged 220/100; in the legs 120/100.

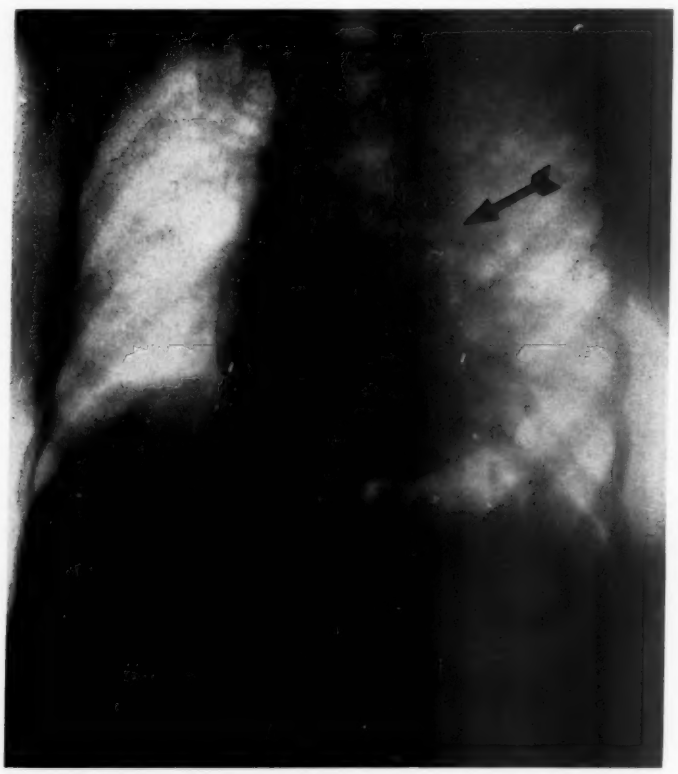

Fig. 4.—Case 2. Left oblique position. The arrow points to coarctation. The left ventricular cavity is small. There appears to be marked hypertrophy of the left ventricular wall. The arteries of the neck are dilated.

Laboratory Findings. Hemoglobin 83 per cent, W.B.C. 8,300, polymorphonuclear leucocytes 21 per cent, monocytes 4 per cent, eosinophilic leucocytes 1 per cent. Urine examination: specific gravity 1.030, albumin faint trace; microscopic, occasional R.B.C. and W.B.C. Blood Chemistry: Urea N, 8 mg., sugar 90 mg., cholesterol 220 mg. per 100 c.c. Blood Wassermann was negative. Basal metabolic rate was +52 per cent and +43 per cent on separate occasions. The patient was given Lugol's solution and the basal metabolic rate fell progressively to +17 per cent. The sleeping pulse rate ranged between 104 and 120 per minute.

Circulation studies: Venous pressure 14 cm.: saccharine time 8 seconds.

Blood flow studies:

Left arm—32° C.—37 c.c./per min. per c.c. of arm volume.
42° C.—60 c.c./per min. per c.c. of arm volume.

Right foot—32° C.— 7.1 c.c. per min. per c.c. of leg volume.
42° C.—22.6 c.c. per min. per c.c. of leg volume.

ECG: Sinus tachycardia, rate 130 per minute. Left axis deviation. QRS of high voltage. R-T depressed slightly in Leads II and III. These evidences indicate left ventricular enlargement.

X-ray examination of the chest: "Heart enlarged to the left; the 4th and 5th and 6th ribs show a slight notching of their inferior surfaces at the postero-axillary line. The lungs show no abnormality. Roentgenkymogram shows marked irregularities in diastole and some diminution in pulsations at the apex and supra-apical area of the left ventricle. Pulsations of the aortic arch are within normal limits. There appears to be some diminution in the pulsations transmitted to the esophagus in the region of the lower thoracic aorta."

Fig. 5.—Case 2. Lateral view. Arrow points to coarctation. The ascending and descending aorta on either side of the coarctation are of normal caliber.

Comment.—The presence of extreme hypertension, cardiac murmurs, lowered blood pressure in the legs, differences of blood flow in the upper and lower extremities, absence of findings pointing to renal disease and the roentgen finding of notched ribs suggested the diagnosis of coarctation of the aorta. However, the history of a normal blood pressure before the onset of the present illness was, to say the least, disturbing. The coexistence of Graves' disease also led to considerable confusion. Contrast visualization was therefore of utmost importance. A narrowed segment of the aorta was demonstrated in the pars descendens distal to the exit of the left subclavian artery. The left ventricle was markedly hypertrophied, the ventricular cavity being relatively small. The supracardiac portion

of the aorta was dilated. The right ventricle was of normal size but the pulmonary artery appeared irregularly dilated.

SUMMARY

1. Contrast visualization of the aorta offers an excellent diagnostic aid in clinically obscure or atypical cases of coarctation of the aorta.

2. The fluorographic multiple exposure technique is especially advantageous, since many photographs with fluoroscopic observation of the required phase of the cardiac cycle are obtained.

3. There has been no undue reaction to the necessary procedures, whether in children or in adults.

REFERENCES

1. Robb, G. P., and Steinberg, I.: Visualization of the Chambers of the Heart, the Pulmonary Circulation and the Great Vessels in Man, A Practical Method, Am. J. Roentgenology and Radiotherapy **41**: 1, 1939.
2. Robb, G. P., and Steinberg, I.: Visualization of the Chambers of the Heart, etc. Methods and Results, J. A. M. A. **114**: 474, 1940.
3. Sussman, M. L., Steinberg, M. F., and Grishman, A.: Multiple Exposure Technique in Contrast Visualization of the Cardiac Chambers and Great Vessels. Accepted for Publication, Am. J. Roentgenology and Radiotherapy.

BALL THROMBI IN THE HEART

Report of Three Cases

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THE first observation of a ball thrombus in the heart was published by William Wood[1] in 1814. The condition is rare, the literature containing reports of 31 cases.[5] Reviews of the literature have been made by Welch[2] (1899), Hewitt[3] (1916), Abramson[4] (1924) and Aronstein and Neuman[5] (1939).

Ball thrombi are so called because of their spherical or ovoid shape and their lack of attachment. The diameter has varied from 1 to 4 cm. and has been greater than that of the orifice of the chamber in which the thrombus lay. The color has been various shades of gray, red and brown and the surface has been either smooth or rough. Some have shown central softening; others were solid, occasionally with lamination. In the authentic cases the ball thrombus has been located in the left atrium and mitral stenosis has been present. These general characteristics are summarized by Welch[2] as follows:

> "(1) entire absence of attachment and consequent free mobility; (2) imprisonment in consequence of excess in the diameter of the thrombus over that of the first narrowing in the circulatory passage ahead of it; and (3) such consistence and shape that the thrombus must not of necessity lodge as an embolus in this passage. The third point does not prejudice the question of the possibility of a ball-thrombus lodging as an embolus; but it excludes from the group . . . detached, shaggy, irregular masses . . . as must necessarily be caught at once as emboli in the narrowed passage in front."

Mitral stenosis is important in the production of ball thrombi in that it is often associated with auricular mural thrombi, portions of which may become detached and then held back by the narrowed orifice. Auricular fibrillation, usually present in cases of ball thrombi, is another important factor in that it favors the formation of mural thrombi and perhaps their detachment. Auricular fibrillation also causes incomplete emptying and a tendency to rotary motion of the blood in the atria, factors which interfere with the lodgement and fixation of thrombotic masses in the valve orifice.

In some instances a rough or projecting spot on the thrombus has indicated its previous point of attachment and also its recent separation. Thus certain ball thrombi have had a spherical shape while still attached. No doubt in other instances irregularly shaped bits of mural thrombi have become detached and have rotated about, growing by

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successive accretions and assuming a spherical shape by a process of moulding and grinding.

The clinical diagnosis of a ball thrombus is difficult. Usually there have been no symptoms suggestive of its presence. In the few cases in which an antemortem diagnosis has been made or considered, there has been widespread and more or less transient interference with the peripheral circulation in a case of mitral stenosis with auricular fibrillation.[6] This phenomenon is presumably due to intermittent partial obstruction of the mitral orifice. Exceptionally, the ball thrombus has caused sudden death. Embolic phenomena have occurred in most of the cases.

Three cases of ball thrombi have been encountered at Cleveland City Hospital in 6,285 consecutive autopsies performed from January 1, 1930, to July 1, 1939, inclusive. This group of autopsies included 156 cases in which rheumatic heart disease was the principal or an important contributory cause of death, an incidence of one case of ball thrombus in 52 autopsy cases of rheumatic heart disease.

CASE 1.—M. L., a white female aged 48, was admitted to Cleveland City Hospital June 24, 1934, for a dilatation and curettage because of symptoms suggestive of a uterine carcinoma. In addition, she had had several "fainting spells" and had become fatigued easily, during the past few years.

General physical examination showed nothing remarkable. The cardiac mechanism was normal except for premature contractions, and no murmurs were heard. There were no signs of myocardial insufficiency and the patient was considered to be a normal risk for anesthesia.

On June 28, a dilatation and curettage was performed. The biopsy material did not show carcinoma. The patient's immediate postoperative course was normal.

On the sixth postoperative day, July 4, 1934, the patient complained of severe pain in the legs. The arteries of the legs could not be felt to pulsate. The cardiac mechanism was now auricular fibrillation. During the following few days, ischemic necrosis of the right foot and the distal portion of the right leg appeared. The left leg was pulseless but did not show ischemic necrosis. The patient died July 8, 1934, the clinical diagnosis being auricular fibrillation of obscure etiology and ischemic necrosis of the right foot and right leg due to an embolus which had lodged at the bifurcation of the aorta causing complete obstruction of the right iliac artery and partial obstruction of the left iliac artery.

Autopsy Findings.—(Autopsy No. A 9611, performed by Dr. D. J. Rehbock, eighteen hours after death.) The heart showed a rheumatic tricuspid stenosis of moderate degree and a severe mitral stenosis, the diameter of the orifice being 1.3 cm. The left auricular appendage was large, and attached to the wall was a thrombus of irregular shape which measured 6 by 2.5 by 2.5 cm. in size. Lying free in the dilated left atrium was a roughly spherical, fairly smooth surfaced thrombus which measured 3 cm. in diameter. Both thrombi were grayish-brown in color and showed central softening and liquefaction.

The distal portion of the aorta was almost completely occluded by an embolus. The right foot and distal part of the right leg showed ischemic necrosis. There were several moderately large infarcts in the spleen and the right kidney.

Microscopically the thrombus attached to the wall of the left auricle was well organized in its deeper portion. The ball thrombus showed a thick peripheral area of dense homogenous basophilic unorganized material. The aorta showed a large, partially depigmented unorganized thrombus attached to the intima.

CASE 2.—S. C., a white female aged 86, entered Cleveland City Hospital April 19, 1935, with a complaint of pain in the right foot of six days' duration. For the past two years the patient had had increasingly frequent attacks of unconsciousness of short duration. Two weeks prior to admission she became orthopneic and noted palpitation. The pain in the right foot had been sudden in onset, constant and severe. The foot had turned a bluish-black color the day of admission.

Examination showed the patient to be dyspneic. There were râles at the bases. The heart was enlarged and the cardiac mechanism was auricular fibrillation with an apical rate of 192 beats per minute. No murmurs were distinguishable. The liver was enlarged. The right foot showed early ischemic necrosis and the right femoral, right popliteal and right dorsal pedis arteries could not be felt to pulsate. The arteries of the left leg could be felt to pulsate. The arteries in general showed evidence of moderate arteriosclerosis.

The patient's condition became poorer and she died May 4, on the fourteenth hospital day. The clinical diagnosis was generalized arteriosclerosis, coronary artery sclerosis, cardiac hypertrophy, myocardial insufficiency, auricular fibrillation, and an embolus in the right femoral artery with ischemic necrosis of the right foot.

Autopsy Findings.—(Autopsy No. A 10008, performed by Dr. G. W. Hobson, six hours after death). The heart showed moderately severe coronary arteriosclerosis and myocardial sclerosis, but in addition there was a severe rheumatic mitral stenosis, the diameter of the orifice being 1.6 cm. The left atrium contained an irregular surfaced, yellowish-pink, soft centered, spherical thrombus which measured 2.2 cm. in diameter. No mural thrombi were present at any point.

The various arteries showed rather severe arteriosclerosis. The right femoral and right popliteal arteries were thrombosed. Whether this was of embolic origin or whether it had originated in situ could not be determined. The right foot showed ischemic necrosis.

CASE 3.—D. B., a white male aged 46, was admitted to Cleveland City Hospital January 24, 1938, with the complaint of "heart trouble." On previous admissions in 1928 and in 1934, the diagnosis was mitral stenosis, auricular fibrillation and a cerebral embolus.

Examination showed the typical signs of mitral stenosis and auricular fibrillation. There was evidence of mild myocardial insufficiency and signs of consolidation in the lower lobe of the left lung, interpreted as infarction.

The patient improved quickly on appropriate treatment and was discharged February 11, 1938. While walking through the ward on his way out of the hospital he fell dead. It was thought that a pulmonary embolus was the cause of death.

Autopsy Findings.—(Autopsy No. A 11999, performed by Dr. T. B. Keller six hours after death.) The heart showed a severe rheumatic mitral stenosis, the orifice averaging 6 mm. in diameter. A fairly smooth surfaced, ovoid shaped thrombus measuring 2 by 2 by 4 cm. was wedged into the mitral orifice so as to cause complete obstruction. In the left auricular appendage there was a short stock-like mural thrombus which measured 5 mm. in diameter. The ball thrombus possessed a pedicle, the structure of which was similar to that of the mural thrombus. In the cavity of the left ventricle there was a small thrombus which had become detached from the ball thrombus.

The other important postmortem findings included recent and remote bilateral infarcts of the lungs, and remote infarcts of the brain, spleen and kidneys.

COMMENT

In Case 1 it is debatable whether or not the ball thrombus caused symptoms. The attacks of syncope may have been due to transient cerebral ischemia caused by temporary obstruction of the mitral orifice, but that point is highly speculative since the patient was not examined during such an attack. The ball thrombus itself was typical in appearance and in the circumstances under which it occurred, i.e., in the left atrium in a case of mitral stenosis with auricular fibrillation.

Again in Case 2 the transient attacks of unconsciousness may or may not have been due to the ball thrombus intermittently obstructing the mitral orifice. The ball thrombus in this case was typical in regard to appearance and the conditions under which it occurred. The patient's age, 86, is noteworthy. The oldest age previously recorded in a case of ball thrombus is 55 years.[5]

In Case 3 the ball thrombus caused the patient's sudden death by wedging into the mitral orifice. In its location and in regard to the circumstances under which it occurred, the thrombus was typical of a ball thrombus.

SUMMARY

Three ball thrombi have been encountered in 156 consecutive autopsy cases in which rheumatic heart disease was the principal or an important contributory cause of death. Rheumatic mitral stenosis with auricular fibrillation was present in all three cases. The ball thrombi occurred in the left atrium. It cannot be determined whether the ball thrombi were the cause of attacks of syncope which two of these patients suffered. The third patient died suddenly. At autopsy a smooth ovoid shaped thrombus was found wedged into the stenosed mitral orifice.

REFERENCES

1. Wood, W.: Edinb. M. & S. J. **10:** 50, 1814.
2. Welch, W. H., in Allbutt, F. C.: A System of Medicine, London, Macmillan Co., 1899, vol. 7, p. 185.
3. Hewitt, J. H.: Free Thrombi and Ball Thrombi in the Heart, Johns Hopkins Hosp. Rep. **17:** 1-80, 1916.
4. Abramson, J. L.: Ball Thrombi of the Heart, Ann. Clin. Med. **3:** 327, 1924.
5. Aronstein, C. G., and Neuman, L.: Ball Thrombus of the Heart: Report of a Case With Review of the Literature, Arch. Path. **27:** 907, 1939.
6. Elson, J.: Free Ball Thrombus of the Left Auricle, AM. HEART J. **10:** 120, 1934.

Department of Clinical Reports

BULLET WOUND OF THE HEART, WITH CORONARY ARTERY LIGATION

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INSTANCES of bullet or knife wounds of a coronary artery, in which the patient is operated upon and survives, are rare enough to justify reporting. Even wounds produced by a needle may prove fatal.[1] The increasing frequency of heart wounds is indicated by the fact that two series of more than twenty cases have been reported by Elkin[2] and Bigger[3] during the past decade. These surgeons, together with Beck,[4] have expounded the principles of diagnosis and treatment.[5]

CASE REPORT

R. W. (U-82423), a 27-year-old colored man, was admitted to the Cincinnati General Hospital at 10:19 P.M. on Nov. 15, 1937, ten minutes after being shot in the chest. He was pulseless and comatose. He smelled of alcohol and at times thrashed about wildly. Examination revealed a sturdy negro in profound shock, with cyanosis and a cold, sweating skin. The respirations were rapid and shallow. There was a bullet wound, almost in the midline, at the level of the fourth intercostal space. The muffled and distant heart sounds could be heard only at the left sternal border, but the rhythm was normal. The cardiac dullness was increased a little at the base. No evidence of pneumothorax or hemothorax was found. The jugular veins stood out like cords. The venous pressure (direct method) was more than 36 cm. of water. The abdomen was soft, but apparently slightly tender below the left costal margin. The patient vomited several times, but the vomitus did not contain blood.

Acacia and saline (500 c.c.) were administered intravenously, and the pulse became palpable. The systolic blood pressure varied from 60 to 80 mm. Hg, and a marked paradoxical pulse was noted both by palpation and auscultation. A roentgenogram showed that the bullet was apparently just beneath the diaphragm. No air could be seen below the diaphragm, but the patient had been lying flat. The heart was not enlarged. Fluoroscopic examination was not attempted.

Because the signs of tamponade indicated that the cardiac condition was more perilous than any possible abdominal injury, his chest was opened. The operation was performed by Dr. Josiah H. Smith. The following is a résumé of his note.

The incision was made under local novocain anesthesia. The third, fourth, and fifth costal cartilages and a portion of the sternum were removed. The left medial reflection of the pleura was pushed laterally. The pericardium was blue, tense, and pulsated very little. Upon opening it, about 600 c.c. of blood gushed out. The systolic blood pressure immediately rose to 110, and the patient recovered consciousness.

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The heart was then delivered into the wound. The anterior descending branch of the left coronary artery had been severed by the bullet, and this accounted for the major portion of the bleeding. A laceration of the heart was found on the anterior surface about 1.5 cm. above the apex, just medial to the severed artery (i.e., to the patient's right). Bleeding was controlled by the finger. The ventricle had not been punctured. A stay suture of medium black silk was placed through the apex of the heart.[4] The bleeding coronary artery was ligated. The rent was closed with three interrupted sutures without disturbing the endocardium. During the manipulation the heart beat became irregular and the systolic blood pressure fell to about 60. This necessitated releasing the traction suture on several occasions, which was followed by restoration of normal rhythm and return of the blood pressure to 110.

The pericardium was loosely closed with interrupted sutures of medium black silk, leaving room for drainage. A small wick was inserted underneath the muscles, but not down to the pericardium. A 150 c.c. transfusion was given during the operation.

Postoperative Course.—The patient stood the operation well, and immediately after it the paradoxical pulse had disappeared. There were occasional premature beats. The abdomen remained negative. An electrocardiogram was taken at 2 A.M. on Nov. 16, less than two hours after the coronary artery was tied.

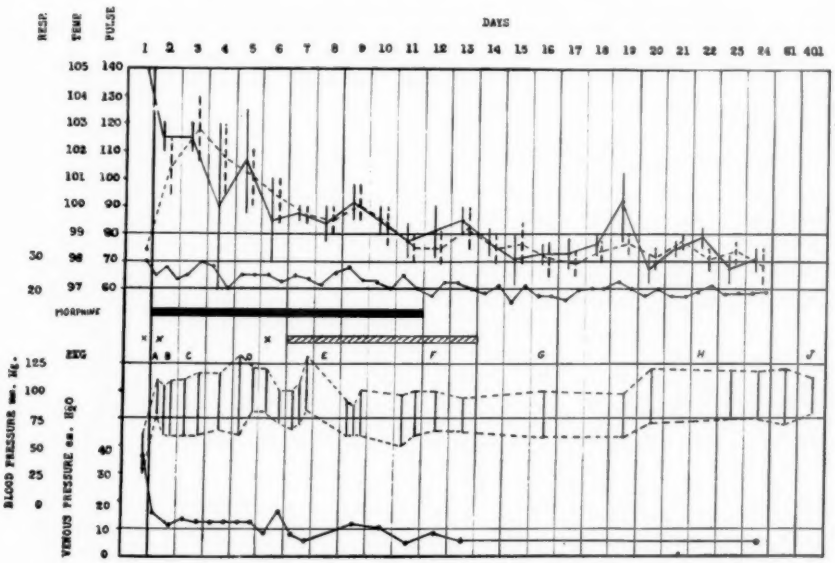

Fig. 1.—Record of the temperature, pulse rate, respiration, arterial blood pressure, and venous pressure, before and after operation. The *X's* indicate transfusions. The letters refer to the electrocardiograms in Fig. 2. The cross-hatched line indicates days when aminophyllin was given. In the temperature (- - -) and pulse (———) curves, the daily fluctuations are recorded and the means are connected. The period of tachycardia on the nineteenth day followed smoking.

The course of events can be followed by referring to the chart (Fig. 1). He was given transfusions of 500 c.c. and 250 c.c., as indicated. Morphine was used to keep him quiet, and, after four stormy days, his condition improved steadily. He experienced no chest pain. No pericardial friction rub was heard and no murmurs appeared, although he was examined repeatedly. When the drain was removed from the wound thirty-six hours after operation, about 30 c.c. of serous fluid spurted

out synchronously with the heart beat. There was some drainage for the next two days. Thereafter the wound healed uneventfully. Pulsus paradoxus was present during the first week. On the sixth postoperative day the administration of aminophyllin was begun; 21 grains were given daily for a week.[6] On the third postoperative day his pulse rate fell suddenly from 120 to 60, but no electrocardiogram was taken. Once, after smoking, he became alarmed because of palpitation and tachycardia. Subsequently, a tracing was made before and after he smoked two cigarettes. The rate increased from 74 to 105 per minute and the number of extrasystoles decreased temporarily, but there were no other changes. Extrasystoles were continuously present after the operation. There were no embolic phenomena.

Subsequent Course.—He was discharged Jan. 28, 1938, ten and one-half weeks after admission, and advised to rest at home. When he was seen in the surgical clinic Feb. 21, his heart was of normal size, as shown by percussion and roentgenologic examination. He had mild dyspnea on exertion, but no edema. He complained of a stinging pain, at times, which started over his heart and radiated down the left arm to the elbow. The pain occurred only when he was lying down, never upon exertion. Olim and Hughes[7] have pointed out that pain may be absent in such cases because of interruption of the periarterial nerve plexus. Since pain does not occur in some cases of acute coronary obstruction and myocardial infarction, especially in negroes,[8] such cases as this throw little light on the mechanism of its production or reasons for its absence. It has been reported in cases of wound of the heart.

The patient was readmitted for study Dec. 10, 1938, thirteen months after the injury. He had rested during February and March, and possibly thereafter, for he was employed on a Works Progress Administration project. He had no dyspnea, edema, pain, or any other trouble, except that occasionally, when quiet, his heart would "jar" him. A soft systolic murmur was heard at the apex. Extrasystoles occurred every three or four beats. Fluoroscopic study revealed that the left ventricle was slightly enlarged. There was no evidence of pericardial adhesions or constriction. The bullet was far anterior, just in front of the extreme left portion of the apex. The vital capacity was 93 per cent of normal (McKesson-Scott), and the circulation time, with 5 c.c. of 20 per cent decholin, was 16 seconds (normal).

When last heard of, in February, 1940, he was doing manual labor for a railroad and had no symptoms or disability.

DISCUSSION

In none of Elkin's, and in only two of Bigger's twenty-five cases, was the heart wound caused by a bullet. One patient with a superficial wound of the right ventricle recovered. In the other, the "bullet had destroyed a rather large segment of the left border of the heart, including anterior, left lateral, and posterior walls of the left ventricle." Victims of such a wound probably rarely obtain any benefit from surgical procedures. Certain ideal circumstances must prevail in order that a patient with a bullet wound may survive. The path must be such that the integrity of no large area of the myocardium is disrupted. Hemorrhage must not be severe, or must be controlled promptly. Other vital organs must escape lethal damage. What the phase of the cardiac cycle may be makes a difference, for a bullet which could kill in diastole might inflict only slight damage if it struck during systole. Similarly, respira-

tion might alter the position of the heart. Such requirements demand nice precision upon the part of a bullet if it is to wound but not kill. Recovery from *bullet* wounds of the heart is rare because of the infrequent conjunction of so many necessary conditions.

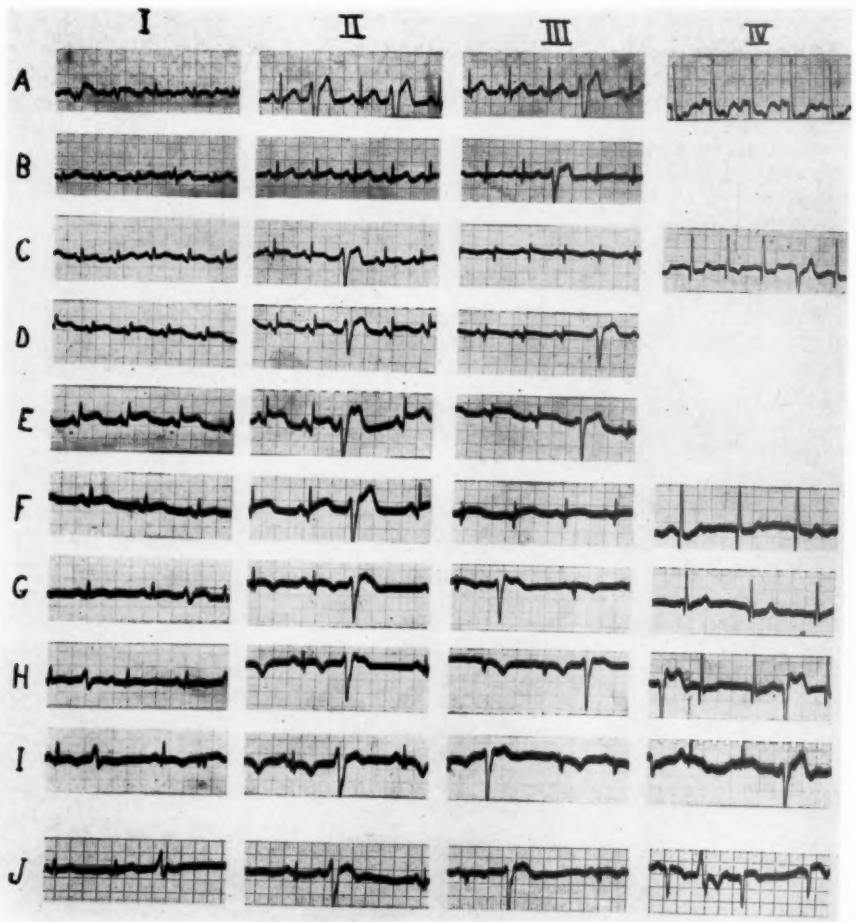

Fig. 2.—Serial electrocardiograms (see also Fig. 1).

Record *A*, taken two hours after operation
Record *B*, ten hours after operation
Record *C*, on the third day
Record *D*, on the fifth day
Record *E*, on the eighth day
Record *F*, on the twelfth day
Record *G*, on the sixteenth day
Record *H*, on the twenty-second day
Record *I*, on the forty-first day
Record *J*, one year and five weeks after operation

Roman numerals indicate leads. In record *J* the Lead IV was taken in accordance with the recommendations of the American Heart Association.[7]

I have found only two cases of heart wounds in which chest leads were made.[7, 9] In one case[9] they were first employed on the fourteenth day after operation, and, in the other,[7] on the thirteenth day. Un-

doubtedly the wound usually is not disturbed during the early postoperative course. Increased movement of the precordium may lead to artifacts (see Record A), as was pointed out by Davenport, Blumenthal, and Cantril.[9] In our case we obtained a chest lead two hours after the operation, on the second day, and regularly after the fourth day. The entire group is not included because it would take too much space, but all of the essential changes are shown in Fig. 2. Unfortunately, the condition of the patient did not permit us to obtain tracings before operation.

ELECTROCARDIOGRAPHIC CHANGES (FIG. 2)

Record *A*, taken two hours after operation, reveals multiple ventricular extrasystoles originating in the region of the apex of the left ventricle. T_1 and T_4 are diphasic, but the T_4 change may have been caused by movement of the precordium.[9] (It is possible that there is a 2:1 A-V block in this lead, with the P waves buried in the T waves.) T_2 and T_3 are high. $S\text{-}T_{1, 2, 3}$ are all distinctly, although slightly, elevated. The axis is normal. A very small Q_1 is present.

Record *B* was taken eight hours after record *A*. The QRS complex in Lead I is splintered. $S\text{-}T_1$ is now clearly elevated, and the T wave has lost its upward convexity. In Lead II a Q wave has appeared, the T waves have decreased in amplitude, and the S-T take-off is more elevated. In Lead III a definite Q wave has appeared, and the S-T take-off is almost isoelectric. The voltage has decreased in all leads.

Record *C*, taken thirty-four hours after operation, shows a high take-off of $S\text{-}T_{1, 2, 3}$ which suggests pericarditis.[10] There is a tendency toward left axis deviation. In Lead IV there is slight depression of the RS-T segment, with an upright T.

Records *D* and *E* show progression of the T-wave changes.

Record *F*, taken twelve days after the operation, shows a lowering of the S-T take-off in Leads I, II, and III and some increase in QRS amplitude; the S-T segment in Lead IV is isoelectric and T_4 is upright.

Record *G*, four days later, shows continued recession of the T-wave changes. The "Pardee" T waves are inverted in the three standard leads. Splintering of the small QRS complexes remains. Q_4 is still present and the T wave is upright.

Records *H* and *I*, taken twenty-two and forty-one days, respectively, after operation, show further progression of changes. Q_1 is a little deeper, and the T waves are sharply inverted and have characteristic conformation.[10] In record *I*, the *Q* wave has almost disappeared in Lead IV.

Record *J*, taken a year and five weeks after operation, reveals that T_1 is upright. T_2 is isoelectric and T_3 is inverted, although not as sharply as a year before. In Lead IV (new method[15]) the T wave is inverted, and corresponds with stage 8 of Bohning and Katz.[11] The Q wave has disappeared entirely. The ventricular extrasystoles persist. A small Q_1 persists.

Interpretation of these electrocardiographic changes presents difficulties. The elevation of the S-T segments in the three standard leads might be accounted for by pericarditis.[10] There was no positive evidence of this clinically; no friction rub was heard, and the most pronounced changes occurred in records *D* and *E*, which were taken on the fourth and fifth days, respectively, when the patient's condition was improving. Wood[12] has emphasized the significance of what he calls the T_2

type of electrocardiogram. In contradistinction to the T_1 and T_3 types of anterior and posterior infarction, this variety is characterized by the fact that it shows the most prominent changes in Lead II. The S-T segment has a higher take-off than in Leads I or III. In Gissane and Schulenburg's case,[13] which Wood describes, there was a penetrating stab wound in the diaphragmatic (posterior), as well as in the anterior wall of the right ventricle, both of which required suture.[14] The three standard leads in our case are very much like others which have been reported.[12, 13] It is possible that the control suture which is placed in the apex to obtain stability[4] produces some damage.

Wolferth and Wood[14] have reported cases of spontaneous coronary occlusion, with anterior and posterior infarction, in which the electrocardiograms were very much like those in this series. Certainly the acute changes include much more than one might expect with uncomplicated, anterior, left ventricular infarction.[7, 9] Over a year after the accident (record *J*), T_2 was isoelectric, and T_3 was still well inverted. This is characteristically the late T-wave position in cases of posterior infarction. The changes in the chest lead were such as one would expect with anterior infarction. In record *J* the new method was used.[15] It shows no Q wave, but a persistent T-wave change (i.e., inversion, which corresponds to an upright T in the antecedent chest leads).

Thus, the T_3 changes that are characteristic of posterior infarction and the chest lead changes which occur as a result of anterior infarction, plus a persistent Q_1, lend some weight to the assumption that both walls of the ventricle were damaged. It may be that a combination of pericarditis, operative trauma, and the bullet itself contributed to the ventricular injury. The amplitude of the QRS complexes has remained low.

The remarkable thing is that, in spite of these persistent changes in the electrocardiogram, the patient should be entirely well. Whether they herald a bad prognosis cannot be stated. The only symptom after the first three months was the annoying palpitation which is so common with extrasystoles. His cardiac function is good enough for heavy physical work.

A search of the literature[2, 3, 5, 7, 9, 12, 13, 16] has revealed no case in which ventricular extrasystoles were a permanent sequel of a direct wound of the heart. In the first lead in record *I* there are extrasystoles from two foci in the ventricle. They usually produced a similar deflection, however, as may be seen in Lead II. Slight differences may be accounted for by superimposition of the P wave. These beats have varied from every other beat to one out of thirty, but usually come every six to eight beats. We have no long strip of tracing which might reveal that they were of parasystolic origin.

SUMMARY

1. Serial electrocardiograms, made after ligation of the anterior descending branch of the left coronary artery, which had been severed by a bullet, are reported. This is the third case in which chest leads were recorded after a heart wound in man.

2. Persistent ventricular extrasystoles and Q- and T-wave changes were observed, and their nature is discussed. The patient is apparently normal, in spite of persistent abnormalities in the electrocardiogram.

I wish to thank Dr. Johnson McGuire for help with the electrocardiograms, and Dr. Josiah Smith for permission to report this case.

REFERENCES

1. Bean, W. B.: Accidental Injury to the Heart by Needle Puncture, New Eng. Med. J. **219**: 257, 1938.
2. Elkin, D. C.: The Diagnosis and Treatment of Wounds of the Heart, J. A. M. A. **111**: 1750, 1938.
3. Bigger, I. A.: The Diagnosis and Treatment of Heart Wounds, South. Med. J. **33**: 6, 1940.
4. Beck, C. S.: Wounds of the Heart; Technique of Suture, Arch. Surg. **13**: 205, 1926.
5. Bigger, I. A.: Heart Wounds, J. Thoracic Surg. **8**: 239, 1939.
6. Fowler, W. M., Hurwitz, H. M., and Smith, F. M.: The Effect of Theophyllin Ethylenediamine on Experimentally Induced Cardiac Infarction in the Dog, Arch. Int. Med. **56**: 1242, 1935.
7. Olim, C. B., and Hughes, J. D.: Stab Wound of the Heart With Coronary Ligation, J. Thoracic Surg. **9**: 99, 1939.
8. Bean, W. B.: Infarction of the Heart II; Symptomatology of Acute Attack, Ann. Int. Med. **11**: 2086, 1938.
9. Davenport, S. L., Blumenthal, B., and Cantril, S.: Electrocardiographic Studies of a Stab Wound of the Heart, J. Thoracic Surg. **5**: 208, 1935.
10. Bellet, S., and McMillan, T. M.: Electrocardiographic Patterns in Acute Pericarditis, Arch. Int. Med. **61**: 381, 1938.
11. Bohning, A., and Katz, L. N.: Four Lead Electrocardiograms in Cases of Recent Coronary Occlusion, Arch. Int. Med. **61**: 241, 1938.
12. Wood, P.: Electrocardiographic Changes of a T_2 Pattern in Pericardial Lesions and in Stab Wounds of the Heart, Lancet **2**: 796, 1937.
13. Gissane, W., and Schulenburg, B.: A Penetrating Stab Wound of the Heart, Lancet **2**: 132, 1937.
14. Wolferth, C. C., and Wood, F. C.: Acute Cardiac Infarction Involving the Anterior and Posterior Surfaces of Left Ventricle, Arch. Int. Med. **56**: 77, 1935.
15. Standardization of Precordial Leads, Supplementary Report, J. A. M. A. **110**: 681, 1938.
16. Koucky, J. D., and Milles, G.: Stab Wound of the Heart, Arch. Int. Med. **56**: 281, 1935.

Department of Reviews and Abstracts

Selected Abstracts

Whittenberger, James L., and Huggins, Charles: Ligation of the Inferior Vena Cava. Arch. Surg. 41: 1334, 1940.

Ligation of the vena cava above both renal veins causes death in dogs in a few hours from surgical shock due to accumulation of blood from two kidneys, in the osterior portion of the body. When oblique ligation of the vena cava between the kidneys is done, allowing development of collateral veins in one kidney, subsequent complete high ligation of the vena cava is well borne; it is not followed by shock or interference with renal function in the kidney with adequate venous drainage.

NAIDE.

Bellet, Samuel, Kershbaum, Alfred, Meade, Richard H., Jr., and Schwartz, Leon: The Effects of Tobacco Smoke and Nicotine on the Normal Heart and in the Presence of Myocardial Damage Produced by Coronary Ligation. Am. J. M. Sc. 201: 40, 1941.

Experiments were performed on normal animals and on animals in various stages of myocardial infarction following coronary ligation in order to study the cardiac effects following the inhalation of tobacco smoke and nicotine injection.

It was found that normal unanesthetized animals were able to tolerate a fairly wide range of dosage with the production of only slight electrocardiographic changes. Following myocardial damage produced by coronary ligation, marked electrocardiographic changes were obtained with a dose that was one-fourth of that required to produce only slight changes in the normal animal. These changes became less marked as the subacute stage was reached and were still less evident in the chronic stage of infarction. However, the electrocardiographic changes after equivalent doses were more marked in the stage of chronic infarction than in the normal controls. The factor of tolerance was considered and was shown not to materially influence these results. Apparently, a parallel existed between the degree of the electrocardiographic effects following the administration of nicotine and the severity of the myocardial damage.

AUTHORS.

Nonidez, José F.: Studies on the Innervation of the Heart. Distribution of the Cardiac Nerves, With Special Reference to the Identification of the Sympathetic and Parasympathetic Postganglionics. Am. J. Anat. 65: 361, 1939.

The present article is based on observations with the chloral hydrate formula of the method of Cajal, carried out on serial sections of the hearts of very young animals (dog, cat). The descriptions refer chiefly to the dog.

Constant differences in the affinity of the nerve fibers for the silver are reported. Most of the deeply stained fibers coursing in the cardiac nerves can be traced to the vagus. Pale fibers of various diameters can be followed into the sympathetic ganglia

which give rami to the heart. They are axones of the sympathetic neurones (sympathetic postganglionics). The preganglionics entering these ganglia stain deeply; the same is true of the preganglionics ending within the intrinsic ganglia of the heart.

In contrast with the sympathetic postganglionics, the axones of the neurones of the ganglia of the cardiac plexus (parasympathetic postganglionics) stain deeply and can be traced to their terminations on the nodes of the conductive system, the arteries and the myocardium of the atria and auricles. The bulk of the parasympathetic postganglionics end on the structures above the coronary sulcus. Few deeply stained fibers are seen in the ventricles, which are supplied chiefly by the sympathetic.

The neurones of the intrinsic cardiac ganglia stain more deeply than those of the sympathetic ganglia.

The ansa subclavia of the dog is described. It conveys afferent fibers of various sizes reaching the middle cervical ganglion in the branches of the middle cardiosympathetic (accelerator) nerve, issued from this ganglion. None of these fibers seem to enter the cervical sympathetic. Small sympathetic ganglia occur in the ansa.

The cardiosympathetic nerves are then considered; of these the superior, issued from the middle cervical ganglion, does not reach the heart proper but ends on the walls of the large arteries. The middle cardiosympathetic (accelerator) nerve is the largest in the dog, and it supplies chiefly the ventricles. The inferior (cardiac branch of the stellate ganglion), when present, carries afferent fibers from diverse areas of the heart; it may run independently or join the accelerator.

The distribution and composition of the cardiovagal nerve differs on the two sides. The right cardiovagal joins the accelerator of its side soon after emerging from the vagus. It sends numerous preganglionics to the ganglia of the deep cardiac plexus, also fibers to the epithelioid bodies (''paraganglia'') between the aorta and the pulmonary artery. The left cardiovagal is an independent nerve which carries the pressoreceptors for the arch of the aorta, base of the left subclavia, and pulmonary bifurcation. It also conveys afferent fibers to the epithelioid bodies and preganglionics to the deep cardiac plexus through a small branch, found in the four puppies examined. Since it receives sympathetic postganglionics from the middle cervical ganglion, it cannot be regarded as a purely vagal branch. Other branches issued from the vagus and recurrent are considered.

The different argyrophilia of the sympathetic and parasympathetic postganglionics, respectively, is discussed. In order to find whether the differences in the impregation of these axones are correlated with similar differences in the neurofibrils of the cell bodies, the necks of dog fetuses (prepared with the Cajal technique) were examined. This study disclosed well-impregnated neurofibrils in the neurones of the nodose ganglion and in parasympathetic neurones occurring in the glossopharyngeal and in the intercarotid (sinus) nerve, respectively. In the neurones of the superior cervical ganglion the neurofibrillar meshwork does not appear impregnated. It is concluded that the differences observed in very young animals result from conditions present in fetal life.

The existence of free nerve endings in the heart is also considered in view of a recent statement by Stöhr in regard to the presence of a neurofibrillar syncytium connecting the vagus and sympathetic fibers. It is pointed out that this ''terminal reticulum'' has not been found by other investigators using the Bielschowsky method and that such a formation, not always impregnated, consists of argyrophil connective tissue fibers. On the basis of the observations reported in the present article, it is concluded that the vagus component of the heart shows a plexiform, but not a syncytial, arrangement and that there are no indications that it merges into meshworks continuous with the sympathetic.

AUTHOR.

Kapp, Frederic, Friedland, C. K., and Landis, E. M.: The Skin Temperature of Hypertensive Rabbits and the Pressor Effects of Heated Kidney Extracts. Am. J. Physiol. **131**: 710, 1941.

The cutaneous vessels in the ears of hypertensive rabbits and normal rabbits responded similarly, both quantitatively and qualitatively, to body warming. The hypertensive state induced by renal ischemia was not associated with measurable diminished peripheral blood flow; this indicated another resemblance between experimental hypertension in animals and the hypertensive state in man.

Heated kidney extracts, injected into hypertensive rabbits, raised the blood pressure to extremely high levels without diminishing skin temperature. Apparently the temporary pressor effect of kidney extract is added to the more permanent hypertension due to renal ischemia, without diminishing peripheral blood flow.

When the same kidney extracts were injected into normal and hypertensive rabbits, the rise in blood pressure was slightly greater in the hypertensive group, except that with large doses in markedly hypertensive animals there appeared to be a maximal value of systolic blood pressure which could not be exceeded. Under these circumstances the blood pressure of the hypertensive rabbits was increased by absolute amounts which were less than those observed with the same extract in normal rabbits.

AUTHORS.

Hueper, W. C.: Experimental Studies in Cardiovascular Pathology. III. Polyvinyl Alcohol Atheromatosis in the Arteries of Dogs. Arch. Path. **31**: 11, 1941.

In dogs repeated intravenous and intraperitoneal injections of a 5 per cent aqueous solution of polyvinyl alcohol result in the development of atheromatous lesions in the aorta and in the carotid and femoral arteries as well as in the appearance of this chemical within the media of smaller arteries and arterioles of the heart and kidney.

The polyvinyl alcohol atheromatosis is morphologically similar to the cholesterol atheromatosis observed in man and rabbits.

The causative mechanism of both types of atheromatosis is represented by a disturbance of the nutritive and oxidative metabolism of the vascular walls brought about by the physicochemical changes prevailing in polyvinylized blood as well as in hyperlipemic conditions.

AUTHOR.

Flexner, James, Bruger, Maurice, and Wright, Irving S.: Experimental Atherosclerosis. II. Effect of Thiamine Hydrochloride and Ascorbic Acid on Experimental Atherosclerosis in Rabbits. Arch. Path. **31**: 82, 1941.

Thiamine hydrochloride and ascorbic acid neither prevent nor accentuate the rise of the blood cholesterol in rabbits fed cholesterol. Furthermore, these vitamins neither inhibit nor enhance cholestero-atherosclerosis of the aorta.

AUTHORS.

Katz, Albert: A Contrast Staining Method for Hemosiderin Pigment in Heart Failure Cells. J. Lab. & Clin. Med. **26**: 700, 1941.

A new staining method here presented allows for a contrast between the heart-failure cells and the surrounding leucocytes. In the sputum, urine, or other material for examination, leucocytes appearing with heart-failure cells are counterstained by Wright's stain, and the hemosiderin takes a bright green stain. The

hemosiderin pigment stands out in sharply contrasted granules against the well-stained basic blue and acidophilic red of the stained leucocytes and other cells.

AUTHOR.

Stewart, Harold J.: Mechanism of Diuresis: Alterations in the Specific Gravity of the Blood Plasma With Onset of Diuresis in Heart Failure. J. Clin. Investigation **20**: 1, 1941.

Change in the level of the specific gravity of the plasma has been used as a measure of change in blood volume, decrease in specific gravity indicating dilution of the blood, and increase in specific gravity, concentration. It appeared that diuresis in the presence of heart failure of the congestive type depended on changes initiated in the tissues, since it was accompanied by decrease in specific gravity of the plasma, that is to say, by dilution of the circulating blood with increase in blood volume. Dilution of the blood preceded the onset of diuresis, and increase in blood volume appeared to be the stimulus that initiated diuretic response of the kidneys. The results were similar not only when diuresis occurred spontaneously, but when it was occasioned by digitalis and by theocalcin and, moreover, whether in the presence of normal sinus rhythm or of auricular fibrillation, and whether the cause of the heart disease was rheumatic fever, syphilis, arteriosclerosis, or hypertension. These studies do not relate to the anatomic portions of the kidney which take part in the accelerated formation of urine in diuresis.

The specific gravity of the plasma must fall from a high level to 1.0255 or lower for the initiation of diuresis. A value below this level may be said to be in the diuretic zone in heart failure, since it corresponds to a dilution of the blood with increase in blood volume of sufficient magnitude to initiate diuresis.

In uncomplicated heart disease the level of specific gravity of the plasma and, by inference, the plasma proteins is in the normal range not only before the onset of failure but during and after recovery from failure. Plasma protein deficiency does not usually participate in the etiology of cardiac edema.

In the technique of taking blood for determinations of plasma or serum proteins or specific gravity of the plasma, samples should not be obtained when diuresis is being initiated or occurring, since low results which do not reflect the usual level for the subject may be recorded. Results should be interpreted in the light of the effect of diuresis which has been demonstrated in this investigation.

AUTHOR.

Adams, Wright, and Sandiford, Irene: The Measurement of Cardiac Output. An Improvement of the Acetylene Method Providing an Inherent Check. J. Clin. Investigation **20**: 87, 1941.

In determining arteriovenous oxygen difference with the acetylene method, multiple samples of "alveolar" and "bag" gas make it possible to identify the establishment of lung-bag equilibrium of the acetylene-air mixture and the beginning of recirculation of acetylene-containing blood to the lungs in each instance. Analytical inaccuracies and leaks, as well as most of the many irregularities encountered in a procedure of this type, are identified.

The time required to achieve lung-bag equilibrium and the time of the first appearance of recirculation are variable, but they usually occur much earlier in the period of rebreathing than Grollman indicated.

The interval between the establishment of equilibrium and the occurrence of recirculation varies, but it is usually long enough to allow at least two complete respirations. This is sufficient for an accurate determination of arteriovenous oxygen difference.

Seventy-six per cent of eighty-two determinations were in error by less than 7.5 per cent.

AUTHORS.

Gross, Desiderio: Oscillometric Studies of the Orthostatic Vasoregulation. Rev. argent. de cardiol. 7: 312, 1940.

Orthostatism induces in normal persons an increase in blood pressure and a reduction of the oscillometric index as measured in the arteries of the lower extremities. In patients with arterial hypertension the increase in blood pressure is also observed but without reduction of the oscillometric index, and in patients with aortic insufficiency there is only moderate reduction of the latter with increase in blood pressure.

The regional increase in blood pressure in the lower extremities and the reduction of the oscillometric index are considered as expressions of regional vasoconstriction induced reflexly by orthostatism as a compensatory mechanism in man. This permanent vasoconstriction may be a determining factor in the pathology of the lower limb arteries.

The great difference in blood pressure between upper and lower extremities which is observed in cases of aortic insufficiency (Hill) is found with equal frequency and intensity in cases of arterial hypertension.

The resistance of the arterial wall has been appreciated indirectly by determinations of mean and diastolic pressure and has been found elevated in cases of hypertension and aortic insufficiency.

The hypothesis is advanced that the increased resistance of the arterial wall, be it due to organic or functional alteration, is the cause of the increased difference of pressure between arms and legs (Hill's sign). The comparison between calculated resistance of the arterial wall and difference between blood pressure measured in arms and that measured in legs lends support to this hypothesis.

AUTHOR.

Waller, John V., Blumgart, Herrman, L., and Volk, Marie C.: Studies of the Blood in Congestive Heart Failure. With Particular Reference to Reticulocytosis, Erythrocyte Fragility, Bilirubinemia, Urobilinogen Excretion, and Changes in Blood Volume. Arch. Int. Med. 66: 1230, 1940.

In the presence of pronounced manifestations of congestive heart failure with increased venous engorgement and elevated pressure, the plasma volume and total red cell mass are increased, the red cell fragility is increased, and the reticulocyte percentage is elevated. The excretion of urobilinogen in the urine and the serum bilirubin concentration are frequently increased, probably as a result of the impaired liver function resulting from anoxemia. As clinical recovery progresses and the total blood volume decreases, the loss in plasma tends at first to be somewhat greater than the decrease in total red cell mass; this results in a slight temporary elevation of the hematocrit. The excretion of urobilinogen in the stools, although at no time significantly increased above the normal values for ambulatory patients, generally shows higher values during the periods of blood destruction than when the evidences of congestive failure become less marked. During the phase of contracting blood volume the reticulocyte percentage decreases, while the values for red cell fragility, serum bilirubin, and urinary urobilinogen become normal.

AUTHORS.

Knox, J. A. C.: The Heart Rate During a Simple Exercise. Brit. Heart J. 2: 289, 1940.

A simple two-step cardiac tolerance test of very brief duration is described. The subject's heartbeats are electrically recorded on a smoked drum throughout the test.

The following indices obtained from the analysis of the records of seventy-five healthy male students are discussed: initial rate, maximum rate, time to reach the maximum rate, acceleration of the heart rate, number of extra beats induced by the exercise, actual and percentage increase on initial rate, and the rate after exercise.

The mean values and variability of these indices are given.

AUTHOR.

Cossio, P., and Yepez, C. Gil: Reduplication of the Second Sound in the Clinical Diagnosis of Bundle-Branch Block. Rev. argent. de cardiol. **7**: 199, 1940.

Heart sounds, electrocardiogram, and venous or arterial pulse were simultaneously recorded in twenty-five patients with bundle-branch block.

Ventricular asynchronism in normal and pathologic conditions is discussed.

The clinical signs of ventricular asynchronism more frequently found were low intensity or reduplication of the first sound, reduplication of the second sound, and, less frequently, a merosystolic murmur.

Reduplication of the second sound is considered the most significant sign of pathologic asynchronism. Its chief characteristic is a great separation between the two components of the split sound, giving rise to a triple rhythm similar to that observed when an opening snap of the mitral valve or a pericardial snap is present. The triple rhythm due to the latter sounds is heard best over the mesocardiac area or at the apex, while that due to the reduplicated second sound is heard best over the second intercostal space.

A relation between duration of the QRS complex and ventricular asynchronism is established.

AUTHORS.

Hoff, H. E., Nahum, L. H., and Kisch, Bruno: Influence of Right and Left Ventricles on the Electrocardiogram. Am. J. Physiol. **131**: 687, 1941.

Extinction of electrical activity of the left ventricle by covering its surface with M/5 KCl permits the recording of the electrogram of the right ventricle, which may be designated as the dextrocardiogram. It is a monophasic-like curve which arises from the R wave and is upright in the three conventional leads.

The levocardiogram is similarly obtained by blocking the electrogram of the right ventricle. It is a monophasic-like wave which arises with the S wave and thus is somewhat later in time than the dextrocardiogram. In all conventional leads it is inverted.

The electrocardiogram represents the algebraic sum of the dextro- and levocardiograms.

Similar results were obtained from dogs, cats, and monkeys.

AUTHORS.

Nahum, L. H., Hoff, H. E., and Kisch, Bruno: The Significance of Displacement of the RS-T Segment. Am. J. Physiol. **131**: 693, 1941.

Elevation of the R-T segment of the electrocardiogram in the dog, cat, and monkey indicates injury to the left ventricle.

Depression of S-T indicates injury to the right ventricle.

When the injury is restricted to a single ventricle, the RS-T interval in all three conventional leads is deflected in the same direction.

Elevation of R-T in one lead and depression of S-T in another indicate that the damage involves contiguous areas of both ventricles.

Elevation in Lead I and depression in Lead III indicate an anterior lesion.

Depression in Lead I and elevation in Lead III indicate an apical or posterior lesion.

Lesions of the anterior surface of the heart are recorded best with the animal on its side, while lesions of the posterior surface of the heart are recorded best with the animal on its back.

AUTHORS.

Hoff, H. E., and Nahum, L. H.: The Factors Determining the Direction of the T Wave: The Effect of Heat and Cold Upon the Dextro- and Levocardiogram. Am. J. Physiol. **131**: 700, 1941.

The T wave results from the interference of the terminal portions of the dextro- and levocardiograms.

Heat curtails the dextro- and levocardiograms.

Cold prolongs the dextro- or levocardiogram.

Prolongation of the dextrocardiogram or shortening of the levocardiogram causes an upright T wave.

Shortening of the dextrocardiogram or lengthening of the levocardiogram inverts the T wave.

AUTHORS.

Mainzer, F., and Krause, M.: The Influence of Fear on the Electrocardiogram. Brit. Heart J. **2**: 221, 1940.

On the operating table immediately before induction of general anesthesia, abnormal electrocardiographic records were found to develop in roughly two-fifths of fifty-three cases, in comparison with the tracings of the previous day. These alterations were observed in persons with cardiac disorders, where they merely accentuated the pathologic character of the cardiogram already existing, and also occurred frequently in patients with normal cardiograms. While in a number of patients the changes disappeared during anesthesia, or at least by the next day, they were in some cases still encountered twenty-four hours after the operation.

The changes may be classified into three groups:

A. S-T is depressed below the isoelectric level, and T is low, inverted, or absent altogether, S-T and T showing some deformation similar to that appearing in coronary insufficiency.

B. P and T are high and become sharply pointed, as in neurocirculatory asthenia.

C. A combination of the changes quoted under A and B.

Factors likely to modify the records, other than the excitement owing to fear of the impending operation, can be ruled out. In some patients the curve returns to its original shape even while they are still under the influence of the anesthetic; this supports the hypothesis of a fear reaction.

In view of the analogies existing between "fear-electrocardiograms" and other types of tracings, it is assumed that the curves of type A are brought about by a reduced coronary flow, mainly to be attributed to vagal stimulation; that sympathetic stimulation is responsible for the development of the curves of type B; and that type C is the result of the interaction of both factors. It is improbable that only humoral factors could be active in bringing about these phenomena, in view of their rapid disappearance.

Thus in clinical cardiography a number of abnormal records that can be explained in no other way probably present genuine fear-tracings, particularly where neurotic persons are concerned.

Death from cardiac failure on the operating table immediately before the induction of general anesthesia, as well as during anesthesia, should, therefore, at least in some cases, be considered as the extreme outcome of an otherwise usual fear-reaction.

The coronary spasms of an ordinary attack of angina pectoris may give rise to the formation of microscopically recognizable necrotic foci in the myocardium. Neurogenic (vagal) lesions of the coronary arteries and myocardium have also been encountered in animal experiments. Thus myocardial damage could be induced by the vasomotor fear-reaction, as becomes apparent in the curves of type A, and could be attributed to coronary constriction of vagal origin.

AUTHORS.

Nyboer, Jan, and Hamilton, James G. M.: Esophageal Electrocardiograms in Auricular Fibrillation. Brit. Heart J. **2**: 263, 1940.

Nine cases of auricular fibrillation have been examined by means of the esophageal lead; in four a return to normal rhythm was observed.

Auricular intrinsic deflections were obtained in five cases, one of paroxysmal fibrillation, two of recent fibrillation responding to quinidine, and two in which the arrhythmia remained established.

Auricular intrinsic deflections were not found in the remaining four cases, one of paroxysmal and three of established fibrillation.

No association was found between the presence of auricular intrinsic deflections in the esophageal cardiograms of the patients studied and the likelihood of reversion to normal rhythm.

The mechanism of auricular fibrillation is discussed in the light of the curves obtained.

AUTHORS.

Vedoya, R., Videla, Gonzalez, and Aguiar, R.: Sino-Auricular Block. Rev. argent. de cardiol. **7**: 277, 1940.

The characteristics of the four cases presented confirm the existence of two principal types of sinoauricular block:

Type I: Progressive prolongation of sinoauricular conduction ending in block of one sinus impulse and absence of auricular and ventricular contraction.

Type II: Occasional cardiac standstill, the interval being approximately double the interval between two normal beats. This type is generally known as "sinus arrest."

The delay with which sinus impulses reach the auricles and ventricles favors the escape of secondary centers and may give rise to two situations:

a. The secondary center commands the whole heart perhaps marking the sinus rhythm.

b. The secondary center commands ventricular activity, while the auricles respond to sinus impulses ("dissociation with interference").

The possibility that these may be other forms of sinoauricular block is discussed, e.g., delayed sinoauricular conduction and complete sinoauricular block.

The mechanism of production of this arrhythmia is discussed. Type I cannot be explained by a disturbance in stimuli formation, but suggests an obstacle in the conduction of the impulse from its point of origin to auricular myocardium.

The expression "sinoauricular block" is adequate for all types of the arrhythmia described and should be used instead of the more common "sinus arrest."

AUTHORS.

Geiger, Clyde J., and Hines, Laurence E.: Prenatal Diagnosis of Complete Congenital Heart Block. J. A. M. A. 115: 2272, 1941.

A case of complete heart block was diagnosed before birth. A sudden slowing of the fetal heart rate which occurred between seventeen and nineteen hours before birth pointed to the onset of the abnormal mechanism at a specific time. The sudden onset of the heart block demanded a differential diagnosis between heart block and intrauterine asphyxia. No anatomic abnormalities in the heart have been demonstrated, and the child is alive and healthy at 5 months of age.

AUTHORS.

Konstam, Geoffrey, and Sinclair, H. M.: Cardiovascular Disturbances Caused by Deficiency of Vitamin B_1. Brit. Heart J. 2: 231, 1940.

Three cases of cardiovascular disturbances caused by deficiency of vitamin B_1 have been reported in greater London.

In two of them diagnosis was confirmed by finding a very low amount of vitamin B_1 in the blood; in the third no vitamin estimation was done.

Two of the patients were chronic alcoholics, and in one of these cirrhosis of the liver was present. The third lived on a very poor diet with a relatively high carbohydrate content. All three suffered from peripheral neuritis.

Achlorhydria or hypochlorhydria was present in all the cases.

The diagnosis, the reaction to treatment, the influence of diet and alcohol, and other associated factors have been discussed.

AUTHORS.

Swan, W. G. A., and Laws, F.: A Case of Beri-Beri Heart. Brit. Heart J. 2: 241, 1940.

A case of beri-beri heart is recorded.

The deficiency was apparently due solely to defective diet.

A clinical cure followed the administration of vitamin B_1.

Electrocardiographic and radiological changes were present but disappeared after vitamin B_1 had been given to the patient.

AUTHORS.

Warburg, Erik: Myocardial and Pericardial Lesions Due to Non-Penetrating Injury. Brit. Heart J. 2: 271, 1940.

Fifty-nine cases of myocardial or pericardial damage due to nonpenetrating blunt injuries have been collected and are reviewed with data from a previous series of 202 similar cases.

The traumatic cardiac lesions described include pericarditis, heart block, myocardial damage, auricular fibrillation and other disturbances of rhythm, and angina pectoris. Auricular fibrillation occurred in one-fifth and angina pectoris in one-fourth of these fifty-nine cases. Seven cases of traumatic coronary thrombosis are cited.

In almost all cases the injury involved the thorax and sometimes the fracture of one or more ribs. However, in a few cases injuries distant from the heart were responsible for cardiac damage.

Arteriosclerosis and hypertension were sometimes present and may have rendered the heart more susceptible to injury from violence to the chest.

AUTHOR.

Wechsler, I. S., and Kaplan, Abraham: Cerebral Abscess (Paradoxic) Accompanying Congenital Heart Disease. Arch. Int. Med. 66: 1282, 1940.

The two cases of cerebral abscess are reported, first, because of the rarity of the condition and, second, because early recognition may possibly lead to successful surgical relief. Twelve cases have been reported in the literature, and the two additional ones bring the total to date up to fourteen. In only three, including our own two, was the condition diagnosed during life and therefore treated surgically; in the rest the anomaly was not even suspected. As a rule a diagnosis of embolism or thrombosis is made. The rapidity with which the abscess generally forms justifies in some measure such a diagnosis. But the presence of fever, leucocytosis, possibly slow pulse, and early fundus changes should draw attention to the presence of an expanding intracranial lesion, more particularly an abscess. In cases in which the diagnosis is doubtful, pneumoencephalographic examination may be indicated. The important point to bear in mind is that abscess of the brain, though rare, can occur in patients with congenital heart disease, even though a primary focus of infection may not be detected. In any event, early recognition as well as familiarity with the complication and with the underlying pathologic changes may lead to more successful surgical intervention.

AUTHORS.

Reindell, H.: Size, Form, and Movement of the Athlete's Heart. Arch. f. Kreislaufforsch. 7: 117, 1940.

This monograph covers the historical background and incorporates the author's own x-ray studies of 116 young athletes 15 to 18 years of age and 421 older athletes. He concludes that in youths there is no enlargement of the heart caused by athletics, although the longitudinal diameter increase indicates a left ventricular hypertrophy. In the course of years, enlargement slowly develops with an increase in transverse diameter. The duration and degree of exertion seem to be related to the rate of development of enlargement. Enlargement may be of the left or of the right ventricle. The enlargement of the athlete's heart must be distinguished from that of disease. Kymography may be useful in differentiation. The athlete's heart enlargement is not evidence of heart disease but is a means whereby the greater effort of the athlete may be more readily accomplished.

KATZ.

Christian, Henry A.: The Determinative Background of Subacute Bacterial Endocarditis. Am. J. M. Sc. 201: 34, 1941.

If in any disease development takes place frequently on a certain background of pre-existing conditions and if this background is recognized, the probability of the existence of the disease in question is enhanced very greatly. The combination of pre-existing conditions may be called the determinative background of a disease.

The determinative background of subacute bacterial endocarditis is (a) rheumatic heart disease, present in 89.33 per cent of 150 consecutive adult cases of subacute bacterial endocarditis caused by the *Streptococcus viridans*, and in 90.24 per cent of 174 consecutive adult cases of subacute bacterial endocarditis of all causes studied at the Peter Bent Brigham Hospital; (b) absence of auricular fibrillation, noted in 2.66 per cent of the *Streptococcus viridans* cases and in 2.87 per cent of cases of all causes; (c) absence of prior cardiac decompensation, noted in fairly marked degree in only 6.66 per cent of the 150 cases and in severe form in only a few cases; (d) youth, young adults, and male sex (61.33 per cent of the 150 cases).

AUTHOR.

Thomson, Scott, and Innes, James: Haemolytic Streptococci in the Cardiac Lesions of Acute Rheumatism. Brit. M. J. 2: 733, 1940.

In conclusion, the authors state that, although at the present time the etiology of rheumatic fever cannot be said to be solved, they are inclined to support the view that the cardiac lesions are caused by infection with hemolytic streptococci.

Hemolytic streptococci were isolated from the hearts of five of ten patients dying of acute rheumatism and never from controls.

Hemolytic streptococci were isolated from damaged valves, but not from undamaged valves.

Other types of streptococci were isolated from both damaged and undamaged valves, but more often from the former.

AUTHORS.

Clahr, Jacob, Klein, Milton D., and Greenstein, Nathan M.: Rheumatic Heart Disease in Pregnant Women. New York State J. Med. 40: 1242, 1940.

The establishment of the prenatal cardiac clinic has led to a threefold increase in the number of diagnosed cases of rheumatic heart disease in pregnancy.

The routine treatment of cases of rheumatic heart disease in pregnancy has been described.

The combination of adequate bed rest and digitalis has been the most effective means in the prevention of intrapartum and post-partum cardiac failure in rheumatic heart disease.

The only maternal deaths occurred in patients who had neither adequate bed rest nor digitalis.

AUTHORS.

Benatt, A., and Taylor, H. J.: The Vascular Response in Chronic Rheumatoid Arthritis. Brit. Heart J. 2: 281, 1940.

Arthritic changes have been seen in cases in which the blood supply to a joint has been restricted.

Investigations have been carried out to find if there might be any functional vascular disturbance in various types and stages of chronic rheumatoid arthritis. The reaction of rheumatic subjects to contrast baths has been compared with that of normal subjects and of subjects suffering from increased vasoconstrictor tone such as Raynaud's syndrome or acrocyanosis.

Unless a marked postural deformity was present, all rheumatic subjects showed a reaction well within normal limits.

Several types of reaction to contrast baths were obtained in cases of so-called Raynaud's syndrome and acrocyanosis. There was no similarity to the reactions obtained in cases of rheumatoid arthritis.

AUTHORS.

Lewis, Thomas: A Note on Pulsating Manubrial Tumour. Brit. Heart J. 2: 260, 1940.

Two cases in which pulsating tumors over the upper part of the sternum in syphilitic subjects seemed to indicate pointing aneurysm are briefly described. Both tumors were secondary deposits from hypernephroma, though in one case aneurysmal dilatation of the aorta was also present. The cases are of interest in emphasizing softness and slowness and a slight but just distinct delay in the rise of the pulse in these pulsating neoplasms.

AUTHOR.

Dill, L. V., and Erickson, C. C.: Effect of Constriction of the Renal Arteries in Pregnancy and in Certain Endocrine States of Rabbits. Arch. Path. **31**: 68, 1941.

Severe renal ischemia in the rabbit produces hypertension, albuminuria, uremia, convulsions, and death. Focal necroses of the myocardium and necroses of the liver occur.

The pregnant rabbit differs from the nonpregnant one in showing increased susceptibility to the effects of renal ischemia or renal injury. The pregnant rabbit demonstrates extensive focal necroses of the liver in a significantly high percentage of experiments.

Renal insufficiency alone or pregnancy alone does not account for the variation of response by the pregnant animal. Other endocrine states do not demonstrate the reaction of the pregnant animal. Hypertension, renal ischemia, or renal injury in association with pregnancy seems to complete the essential combination of factors.

These experimental observations support the thesis suggested in our report of preliminary observations that renal ischemia or renal injury in the pregnant animal produces a syndrome with involvement of physiologic factors similar to, and production of pathologic lesions analogous to, those in at least some of the human toxemias of pregnancy.

AUTHORS.

Nesbit, Reed M., and Ratliff, Rigdon K.: Hypertension Associated With Unilateral Renal Disease. J. A. M. A. **116**: 194, 1941.

Experimental evidence and clinical observations have shown that hypertension may result from pathologic conditions of the kidney which may be bilateral or unilateral.

Three different types of unilateral renal lesions are most commonly associated with hypertension: (1) those produced by gross vascular occlusion of the renal arteries including trauma to the kidney, (2) the obstructive uropathies, and (3) chronic inflammatory lesions.

Since chronic infection appears to be the most important etiologic factor, it would appear that the best treatment of this type is prophylactic. In this regard the value of modern chemotherapeutic methods for the prevention of chronic infections of the urinary tract cannot be overemphasized.

Hypertensive patients should be submitted to complete urologic study as a part of the routine examination, even in the absence of a history of renal disorders or urinary findings suggesting disease of the urinary tract.

The rational treatment of the hypertensive patient with unilateral nephropathy is removal of the diseased kidney, provided the function of the opposite kidney is not significantly reduced. With this restriction, a reasonable expectancy of improvement or cure can be hoped for in the majority of cases.

NAIDE.

Howard, T. L., Forbes, R. P., and Lipscomb, W. R.: Aneurysm of the Left Renal Artery in a Child 5 Years Old With Persistent Hypertension. J. Urol. **44**: 808, 1940.

A case of persistent hypertension as a result of a multilocular aneurysm of the left renal artery in a 5-year-old child is reported. The hypertension was relieved by nephrectomy.

NAIDE.

Sprague, P. H.: A Case of Temporal Arteritis (Horton-Magath Syndrome). Canad. M. A. J. 43: 562, 1940.

A case of arteritis of the temporal arteries is reported. This entity was first described by Horton, Magath, and Brown. The condition, in the patient reported, simulated temporal neuralgia and temporomandibular arthralgia before the correct diagnosis was made. The clinical features of the syndrome are its incidence in the fifth and sixth decades, the prolonged febrile course, loss of weight, anemia, anorexia, and weakness. It must be differentiated from periarteritis nodosa, thromboangiitis obliterans, and rheumatic arteritis. The arteritis is probably not limited to the temporal arteries but may involve other arteries as well.

NAIDE.

Mathe, Charles Pierre: Thromboangiitis Obliterans (Buerger's Disease) of the Spermatic Arteries: Report of a Case. J. Urol. 44: 768, 1940.

A case of thromboangiitis obliterans of the spermatic arteries which was treated by orchidectomy is reported in detail.

Pathologic examination revealed thrombosis of the spermatic arteries and anemic infarct of the central segment of the testis. The pathologic changes encountered in the walls of the spermatic vessels and in the perivascular tissues, as well as the nature of the thrombus, were characteristic of Buerger's disease.

NAIDE.

Griffiths, D. L.: Volkmann's Ischemic Contracture. Brit. J. Surg. 28: 239, 1940.

Observations upon thirty-two cases support the theory that Volkmann's ischemic contracture is due to arterial injury and to the accompanying spasm of the collateral circulation. Clinical signs of arterial occlusion in acute and chronic cases occur regularly, and the histology is shown to be that of muscle infarction. The idea that the contracture results from venous occlusion is rejected because of the absence of any venous compression by hematoma in acute cases, the discovery of arterial lesions in operations at all stages, the occurrence of the contracture after arterial embolectomy, and the production of the lesion in a small series of rabbits by arterial ligature.

The early diagnosis of the lesion is discussed, and its prevention by operations designed to relieve arterial obstruction and collateral spasm is described. Preventive treatment must be initiated early, since the fate of the infarcted muscle is decided by hours and not days. Any case showing absence or weakness of radial pulse following a limb fracture is in danger of developing the Volkmann syndrome.

NAIDE.

Edwards, Edward A., Hamilton, James B., Duntley, S. Quimby, and Hubert, Gilbert: Cutaneous Vascular and Pigmentary Changes in Castrate and Eunuchoid Men. Endocrinology 28: 119, 1941.

With the aid of the Hardy recording spectrophotometer, studies of the cutaneous vascularity and pigmentation of four human male castrates and five eunuchoid patients were carried out before, during, and after treatment with testosterone propionate. In the castrates there was a generally diminished quantity of hemoglobin in the skin, with a higher than normal proportion in the reduced state. This is interpreted as an indication of a smaller cutaneous vascular bed than normal with less blood flow through the region. Administration of testosterone propionate reversed these changes.

Carotene was present in excessive quantity in the skin of untreated castrates but was reduced to normal levels by administration of testosterone propionate.

The eunuchoids showed less deviation from normal than the castrates.

SCHWARTZ.

Dickens, Karl L.: Pulmonary Stenosis Produced by Aneurysm of the Ascending Aorta. Brit. Heart J. 2: 247, 1940.

A case is reported in which an aneurysm of the aorta was so located as to produce pulmonary stenosis and insufficiency and a clinical picture suggestive of aneurysm of the pulmonary artery. Previous reports are briefly reviewed, and it is pointed out that the location of the aneurysm in the reported case is very unusual, as are its ultimate effects upon the pulmonary system.

AUTHOR.

Loeffler, Louis: Pulmonary Embolism and Infarction. Arch. Path. 31: 93, 1941.

In guinea pigs and rats a string tied moderately tightly around a lobe of the lung produced hemorrhagic infarction. The tying of a string suddenly and tightly around a lobe produced anemic necrosis.

Stoppage of the blood supply to the kidneys, liver, lungs, and intestines, to summarize the present and previous experiments, produced anemic necrosis, an observation that might be applied to any organ.

A hemorrhagic infarct, wherever it occurs, requires a continuous blood current and increased venous pressure. The continuous blood flow is maintained either by collaterals, as in the lungs, or by incomplete occlusion of the arteries, as in the intestines.

Occlusion of the pulmonary artery at any point of its course does not, as a rule, cause serious disturbance of the lung tissue, because there is sufficient collateral circulation through the bronchial arteries. The anemic and the hemorrhagic infarcts in lungs represent exceptions to this rule, due to special conditions.

AUTHOR.

Mason, David G.: Subacute Cor Pulmonale. Arch. Int. Med. 66: 1221, 1940.

An example of a case of subacute cor pulmonale is presented. Death was caused by fairly rapid obstruction (clinically recognized twenty-seven days) of the pulmonary arterioles and pre-arteriolar arteries, with ensuing failure of the right side of the heart. The vascular obstruction was due to emboli of small carcinoma cells arising from a primary carcinoma of the breast, for which radical mastectomy had been performed nineteen months previously.

AUTHOR.

Wilkinson, K. D.: Aneurysmal Dilatation of the Pulmonary Artery. Brit. Heart J. 2: 255, 1941.

A case is reported in which an aneurysmal dilatation of the pulmonary artery, due to a congenital defect, led to sudden death from rupture of the walls of the artery, the rupture also producing some degree of dissection of the coats of the artery.

AUTHOR.

Heller, Richard E.: The Pathological Physiology of Varicose Veins. Surg. Gynec. & Obst. 71: 566, 1940.

This is an excellent review of the normal venous circulation and the physiology of varicose veins.

NAIDE.

Dougherty, John, and Homans, John: Venography, a Clinical Study. Surg. Gynec. & Obst. **71**: 697, 1940.

An experimental study in venography is presented in which fifteen cases, six normal and nine abnormal, were studied. The study was limited to the lower extremities and the adjacent portions of the body and was principally concerned with thrombosis.

A 50 per cent solution of an organic iodide commercially known as diodrast (compound) was used in conjunction with an infusion set-up containing physiologic saline solution. In none of the cases was there evidence of reaction.

Two cases are illustrated by photographs of the venograms. The first was a case of acute femoro-iliac thrombophlebitis accompanied by arterial spasm. The second was a case of traumatic femoro-iliac thrombosis, subsequently canalized and complicated by recurrent small embolisms. For comparison, a normal venogram is presented. The literature, both foreign and local, is very briefly reviewed.

NAIDE.

Keen, J. A.: A Case of Complete Obstruction of the Inferior Vena Cava. Brit. M. J. **2**: 823, 1940.

Complete obstruction of the inferior vena cava was discovered accidentally in a dissecting-room subject, a colored man, aged 71 years. There was nothing in the clinical history which gave any explanation of the causation of the venous thrombosis. The man was blind and spent the last eleven years of his life at the Capetown Infirmary. He was not bedridden and could get about for walks. His last illness was a rather sudden collapse with pulmonary congestion; nothing abnormal was found in the abdomen or urine during the last few days before his death.

A short account is given of the anatomy of a case that demonstrates the remarkable potentialities of the body for establishing circulatory anastomoses.

From this case it appears to be possible to establish a complete collateral circulation for the drainage of both kidneys.

AUTHOR.

Hegglin, R.: The Circulation in Disturbed Sugar Metabolism, Especially in Diabetic Coma. Arch. f. Kreislaufforsch. **7**: 1, 1940.

In ten cases of diabetic coma, the occurrence of an S-T depression in Leads I and II and flattening of T in these leads was found, as well as lengthening of electrical systole. Insulin therapy exaggerated these changes. Mechanical systole, measured from heart sound records, was abbreviated in diabetic coma.

KATZ.

Bowers, Warner F., and Kennedy, John C.: Improved Management of Gangrene of the Foot. Am. J. Surg. **50**: 573, 1940.

The authors advise débridement as a preliminary to major amputation in patients with arteriosclerotic gangrene with a septic type of temperature curve. A major amputation is performed after the temperature has been down for two days.

NAIDE.

Book Reviews

THE PHARMACOLOGICAL BASIS OF THERAPEUTICS: By Louis Goodman, M.D., Assistant Professor of Pharmacology and Toxicology, Yale University School of Medicine, and Alfred Gilman, Ph.D., Assistant Professor of Pharmacology and Toxicology, Yale University School of Medicine. 1941, The Macmillan Company, New York, 1383 pages, 126 figures. $12.50.

This book differs from other manuals of pharmacology in that the action of drugs on healthy persons and on the sick is treated far more extensively. It is the best compilation available concerning the action of drugs, as judged by observations made on man.

ISAAC STARR.

THE ELECTROCARDIOGRAM AND X-RAY CONFIGURATION OF THE HEART: By Arthur M. Master, M.D., Associate in Medicine and Chief of the Cardiographic Laboratory, Mt. Sinai Hospital, New York; Associate in Medicine, College of Physicians and Surgeons, Columbia University. Lea and Febiger, Philadelphia, 1939, 222 pages, 71 illustrations, $6.50.

This monograph presents a correlation between roentgenographic and electrocardiographic observations, first, on the normal heart, with particular reference to age, position of the body, habitus, obesity, and pregnancy, and, second, on heart disease caused by hypertension, valvular lesions, congenital malformations, pericarditis, metabolic disturbances, pulmonary disease, and deformities of the chest. The material is presented in atlas form; the roentgenograms and electrocardiograms are side by side, with ample text and legends. As far as a correlation between the two methods is possible, it has been brought out in a very useful and good didactic manner. One is grateful that the author, as a real clinician, presents the clinical background in each case. This may help to combat the unfortunate tendency to render diagnostic service by excessive specialization, such as "reading" electrocardiograms or roentgenograms. The material should be quite valuable, particularly for the beginner.

It is only natural that reviewers should not always be in full agreement with authors, and, in submitting this criticism, it is hoped that some of it will seem acceptable to the author, and perhaps be incorporated in a second edition. In order to give readers of the monograph an opportunity to form their own opinions, it seemed wise to refer specifically to each instance in which a divergent opinion is held.

With respect to the electrocardiograms.—Fig. 21 illustrates right axis deviation as it occurs with the vertical type of heart. It may simulate that of mitral valve disease, but there is not much danger of confusing them because the electrocardiogram which is shown reveals a predominance of P_2 and P_3, whereas with mitral disease, if stenosis prevails, predominance of P_1 and P_2 is observed as a rule. This, for instance, is demonstrated in Fig. 42. Figs. 28 and 44 are accompanied by a statement that a small initial positive deflection in the chest lead (leading from the precordium just within the apex, and from the left leg) is frequently seen when the heart is large. This is noted, of course, when the exploring electrode is placed over the right ventricle; the intrinsic deflection would be high, or higher, if the electrode were placed further to the left, and the lesson is that taking one chest

lead only may give insufficient information. Fig. 28, as well as others, depicts the electrocardiogram which is typical of hypertension, and reference is made to the normal duration of the QRS complex. To point out that, with high voltage, the area of the QRS complex is increased, and that this factor influences the direction of the T deflection, might have been useful. Fig. 30 is said to show a deep Q_4, which is interpreted as indicating involvement of the myocardium; this wave is really S_4. The same wave, which is supposed to be indicative of myocardial damage in Fig. 30, is interpreted in Fig. 28 as evidence of cardiac enlargement. Fig. 34 is said to illustrate marked left axis deviation and high voltage of QRS as a result of ventricular enlargement. The correct interpretation is that a relatively low R_1, when it is associated with a deep S_2 and S_3, is indicative of anterior infarction, usually with aneurysm of the heart, which the author properly presents as such. The legend of Fig. 35 states that slight left axis deviation indicates slight left ventricular enlargement. This is certainly unacceptable. In connection with Fig. 39 there is no mention of prolongation of the Q-T segment. This case, by the way, demonstrates nicely that high voltage, with left axis deviation and normal T deflections in Leads I, II, and IV, may be associated with a marked degree of left ventricular enlargement, caused, in this instance, by aortic regurgitation of syphilitic origin. Fig. 40, from a patient with aneurysm of the aorta and no cardiac enlargement, is accompanied by the statement that the electrocardiogram is entirely normal because the size, shape, and position of the heart are normal. Another reason, however, might be that there was no encroachment on the coronary ostia. In several illustrations, inversion of T_2 and T_3 is interpreted as evidence of right ventricular enlargement. In the case of Fig. 44, however, it is quite certain that this was caused by digitalis, judging from the concavity of S-T in Lead I and the maximal depression of S-T in Lead II. Figs. 45, 54, and 56 reveal a high R_3 with right axis deviation. In connection with Figs. 45 and 54, the author says that high voltage of QRS is associated with enlargement of the left ventricle, but, as a matter of fact, it is also noted with right ventricular enlargement. Obviously, difficulties will arise with respect to both Figs. 54 and 56; left ventricular hypertrophy is out of the question in these cases because of the type of congenital malformation which was present. In connection with Fig. 56, the author speaks of high voltage of QRS in association with an increase in the muscle mass of the heart. Fig. 46 is said to show widening of the P waves, but this is not apparent. The description of Fig. 57 states that its slurred and abnormally long QRS complexes indicate myocardial disease, but this record really shows left bundle branch block. Fig. 58 depicts the electrocardiogram in a case of dextrocardia. The illustration is not well chosen, first, because the voltage of QRS in Lead II is larger than in Lead III, and the reverse holds true for the electrocardiogram in dextrocardia as a part of complete inversion, and, second, because of the diphasic character of QRS. The latter is indicative of an associated congenital anomaly, the existence of which, by the way, is further evidenced by the cardiac enlargement shown in the roentgenogram. Fig. 59 shows high P deflections after recent cardiac infarction, and it is pointed out that the P deflections returned to normal within several weeks. This is correct, but it is also interesting to note that simultaneously the voltage of QRS diminished greatly. Fig. 62 shows a negative P_2 and P_3 in a case of chronic bronchitis and emphysema, and this is regarded as evidence of displacement and rotation of the heart. The accompanying roentgenogram does not reveal displacement, and if the cardiac rotation was the result of low position of the diaphragm, why should P become negative in Leads II and III? The direction of the P deflection, in conjunction with a P-R interval of 0.11 second, makes this an obvious case of nodal rhythm. It would have been more instructive to present a case in which P_2 and P_3 were high; this is common, although not constant, with chronic "cor pulmonale."

With respect to the roentgenograms.—Two basic premises are not fulfilled. First, with very few exceptions, only anterior views are presented, and this limits one's ability to detect cardiac enlargement, especially enlargement of individual chambers. Right ventricular enlargement may thus escape notice; and this explains, for instance, why the author on page 183 states that in uncomplicated emphysema the heart is usually of normal size, although on page 182 he cites the excellent and correct observations of Parkinson and Hoyle, who came to opposite conclusions. Second, it is stated that the roentgenograms were taken at the end of moderate inspiration. As a matter of fact, many of them show that they were made during deep inspiration. This is unfortunate because inspiration causes an unphysiologic position, and hence a morphologically distorted roentgenographic appearance of the cardiovascular silhouette is obtained; also, the roentgenograms are correlated with electrocardiograms which, as a rule, are not taken during deep inspiration. Figs. 1 and 4 are supposed to show normal positions, but actually this is not the case; the narrow shape in Fig. 22 is exaggerated, and Fig. 32 seems to show that the size of the heart is normal (case of long-standing hypertension). Fig. 21 is supposed to illustrate a long, narrow heart, with rotation of the right ventricle forward, but the right ventricle cannot come to lie farther forward than it naturally does. Figs. 7, 8, 23, and 24 are supposed to reveal left ventricular enlargement, but this is not apparent. The roentgenogram shown in Fig. 35 is said to be typical of aortic insufficiency. Such a diagnosis could hardly be made without studying the pulsations fluoroscopically. Page 118 carries the statement that aortic stenosis produces left ventricular enlargement; this is not necessarily true, for hypertrophy, per se, need not cause appreciable enlargement. Fig. 46 shows the cardiac shadow reaching the right side of the costal cage, and this is ascribed to huge enlargement of the right auricle. Actually, it is caused by so-called aneurysmal dilatation of the left atrium. On page 153 it is stated that patency of the foramen ovale brings about enlargement of the auricles. This is not the case. Fig. 54 is said to show that a sharp convexity of the right border of the heart indicates enlargement of the right ventricle. As a matter of fact, right ventricular enlargement does not produce such a contour.

Finally, a few suggestions are submitted with respect to nomenclature, namely, roentgenogram instead of x-ray film, extrapericardial fat instead of pericardial fat, conus of the right ventricle instead of pulmonary conus, atrium instead of auricle (unless used in connection with the term appendage), postural circulatory deficiency instead of neurocirculatory asthenia syndrome. The terms cardiac infarction and coronary occlusion appear to have been used somewhat promiscuously.

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Books Received

LA INSUFICIENCIA CARDIACA: By Dr. Cristián Cortés Lladó, Antiguo Jefe del Servicio de Cardiologia de Cataluña. De la Escuela de Medicina de Barcelona. Compañia General Editora, S. A., Mexico, D. F., 1940, 186 pages, 30 illustrations.

THE VASOMOTOR SYSTEM IN ANOXIA AND ASPHYXIA: By Ernst Gellhorn, M.D., Ph.D., Professor of Physiology, University of Illinois, and Edward H. Lambert, M.D. University of Illinois Press, Urbana, Ill., 1939, 71 pages, 21 illustrations, $1.50.

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